OCULAR INJURIES IN ROAD TRAFFIC ACCIDENTS - A STUDY

Dissertation submitted to

THE TAMILNADU DR.M.G.R.MEDICAL UNIVERSITY
CHENNAI – 600 032.

In partial fulfillment of the regulations
for the award of the degree of

M.S.DEGREE - OPHTHALMOLOGY

STANLEY MEDICAL COLLEGE,

CHENNAI – 600 001.

APRIL 2012

CERTIFICATE

This is to certify that the dissertation entitled **"OCULAR INJURIES IN ROAD TRAFFIC ACCIDENTS - A STUDY"** presented here is the bonafide original work done by **Dr.T.KAVITHA,** in the Department of Ophthalmology, Government Stanley Medical College, Chennai – 600 001, in partial fulfillment of the regulations for the **M.S. DEGREE OPHTHALMOLOGY** Examination of the Tamil Nadu Dr.MGR Medical University to be held in April 2012.

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DECLARATION

I, Dr.T.KAVITHA, solemnly declare that this dissertation entitled, **"OCULAR INJURIES IN ROAD TRAFFIC ACCIDENTS - A STUDY"** is a bonafide work done by me, at Government Stanley Medical College, Chennai between 2009 – 2012, under the guidance and supervision of **Prof.Dr.K.Basker, M.S.,D.O.,** Head of the Department, Department of Ophthalmology.

This dissertation is submitted to Tamilnadu Dr.M.G.R. Medical University, towards partial fulfillment of regulation for the award of M.S.Degree Ophthalmology.

Place: Chennai
Date: **(Dr.T.KAVITHA)**

ACKNOWLEDGEMENT

I express my sincere thanks to **Dr.S.Geethalakhsmi, M.D., Ph.D.,** Dean, Stanley Medical College for permitting me to conduct this study.

I express my profound gratitude to **Prof.Dr.K.Basker, M.S., D.O.,** Head of Department of Ophthalmology for assigning me this interesting topic, guiding me with this vast knowledge and experience and providing me all the necessary facilities.

I am very grateful to **Prof.K.Kanmani, M.S., D.O., Prof.Thangarani, M.S., D.O.,** for their continuous support and guidance.

I am very grateful to my Assistant Professors **Dr.B.Meenakshi, M.S., Dr.P.Geetha, M.S., D.O., Dr.Vinayagamoorthy, M.S., Dr.Nandhini M.S., Dr.Venkatesh, M.S.,** for rendering their valuable advice and guidance for the study.

I wish to express my sincere thanks to all my colleagues who had helped me in bringing out this study.

Finally, I am deeply indebted to all my patients for their sincere cooperation for completion of this study.

CONTENTS

INTRODUCTION

ROAD TRAFFIC ACCIDENTS are common occurrence every day. With ever increasing new drivers and new vehicles, RTAs keep on increasing causing mild to severe injuries to eyes, the special organ designed for vision.

Eyeball is anatomically well protected inside bony orbit socket and orbital margin, covered anteriorly by eye lids with lashes, embedded in cushion of retro bulbar pad of fat behind. Inspite of all these protection it is vulnerable to injuries.

Ocular trauma is single most important cause of monocular blindness Worldwide and second leading cause of visual impairment and blindness in all age groups.

Eye injuries not only results in defective or loss of vision but creates enormous loss to both victim and society productivity

Eye trauma is an evolving sub specialty of its own. The recent breakthrough in understanding mechanisms of injury has dramatically improved the management of eye injuries.

A lot of research has been done in the past, regarding blunt or penetrating injuries of eye. But surprisingly very few datas are available

regarding road traffic accidents and ocular injuries even though it is a common occurrence in daily outpatient department in a multi specialty hospital like our hospital.

So it is of great relevance to create a data regarding RTA and ocular injuries and its effects in a multi specialty hospital like Stanley medical college and hospital located in a heavily populated place like north madras surrounded by lot of industries.

REVIEW OF LITERATURE

OCULAR TRAUMA.

THE BIRMINGHAM EYE TRAUMA TERMINOLOGY (BETT)

OPEN GLOBE-full thickness eye wall wound present

CLOSEDGLOBE-no full thickness eye wall defect present

LACERATING INJURY –open globe resulting from sharp force

PENETRATING INJURY-entrance wound, no exit wound

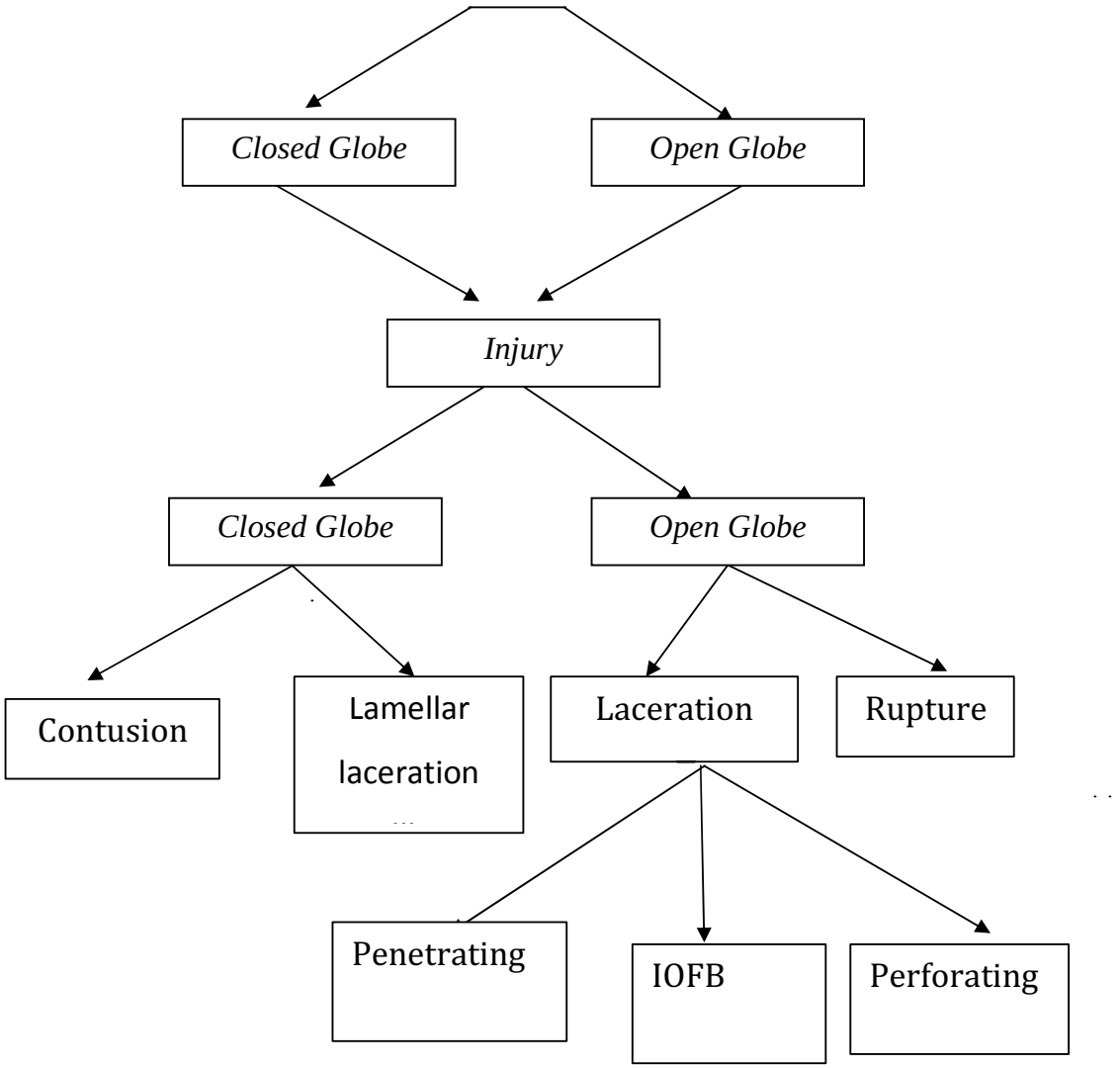
PERFORATING INJURY-entrance and exit wound

INTRA OCULAR FOREIGN BODY-entrance wound with foreign body in eye

CONTUSION INJURY-blunt injury, no open globe

LAMELLAR LACERATION –sharp force, partial thickness eye wall defect.

Eye Injury



SYSTEM FOR CLASSIFYING OPEN GLOBE INJURY:

TYPE: A. rupture

B. penetrating

C. Intra ocular foreign body

D. perforating

E. mixed

GRADE: (VISUAL ACUITY): A.>/=**20/40**

B.20/50 to 20/100

C.19/100 to 5/200

D.4/200 to PL

E.NO PL

PUPIL: (RAPD) Present

Absent

ZONE:

1. Isolated to cornea.
2. Corneo scleral limbus to 5mm posterior to sclera.
3. Posterior to the anterior 5mm of sclera.

SYSTEM FOR CLASSIFING CLOSED GLOBE INJURY

TYPE (mechanism of injury): A.Contusion

B.Lamellar laceration

C. Superficial foreign body

D. Mixed

GRADE (presenting visual acuity):A.>20/40

B.20/40-20/100

C.19/100-5/200

D.4/200- light perception

E. no light perception

PUPIL (RAPD): Present

Absent

ZONE:

1. External (bulbar conjunctiva, sclera, cornea)
2. Anterior (all structures in relation with anterior chamber & pars plicata)
3. Posterior segment (all internal structures posterior to posterior lens capsule)

CALCULATING THE OCULAR TRAUMA SCORE

STEP1: IDENTIFY RELEVANT VARIABLE:

VARIABLE	RAW POINT
INITIAL VISION	
No PL	60
PL/ HM	70
1/200-19/200	80
20/200-20/50	90
>20/40	100
RUPTURE	-23
ENDOPHTHALMITIS	-17
PERFORATING INJURY	-14
RETINAL DETACHMENT	-11
APD	-10

STEP 2: CALCULATE THE SUM OF RAW POINTS=

STEP 3: CONVERT RAW POINTS INTO % CHANCE OF VISUAL OUTCOME

RAW SUM	OTS	NO PL	PL/HM	1/200-19/200	20/200-20/50	>20/40
0-44	1	74%	15%	7%	3%	1%
45-65	2	27%	26%	18%	15%	15%
66-80	3	2%	11%	15%	31%	41%
81-91	4	1%	2%	3%	22%	73%
92-100	5	0%	1%	1%	5%	94%

GENERAL EVALUATION OF TRAUMATIC EYE

An accurate and thorough evaluation is important in eye injury. There should not be any rigid approach but overall examination is adjusted according to history from patient or witness. The status of fellow eye should be noted.

Appropriate sedation, adequate analgesia and protection from further injuries are important during examination. Associated life threatening injuries should be detected and treated accordingly.

SYSTEMIC EVALUATION:

Vital signs and mental status should be checked. Brief assessment of obvious bony or soft tissue injuries made. Once the patient is stable, detailed history and ocular evaluation is done.

HISTORY:

Injuries sustained in high velocity such as motor vehicle accidents are associated with extra ocular injuries which demand immediate attention.

The details of events preceding and leading to injury, description of mechanism of injury, presence of safety glasses, seatbelts, air bags, obtained.

Time of accident and time of coming to hospital are important in both evaluation and treatment of ocular trauma and chemical injuries.

Current and previous medical history medications allergies, past ocular history are obtained. Status of tetanus immunization determined.

Involvement of blunt force may indicate presence of ocular rupture. Place of occurrence of injury affect the outcome of injury.

History of previous ocular surgery may affect the surgical approach such as laceration or rupture that extends near scleral buckle or glaucoma filtering device. Patients who have significant glaucomatous optic atrophy tolerate the post operative pressure elevation poorly. Patients on immune suppression, diabetes or vitamin or protein deficiency may exhibit poor wound healing.

The status of other eye is important in decision making. Patient with normal fellow eye may justify different treatment than one eyed patient.

INITIAL OCULAR EXAMINATION:

VISUAL ACUITY:

Initial visual acuity is the best predictor of final visual acuity. Pinhole acuity is obtained if possible by Snellen chart or ETDRS chart. Tumbling E chart for illiterates and if vision (vn) is very less, counting fingers, hand movements, light perception and projection of rays checked.

CONFRONTATIONAL VISUAL FIELDS:

Static finger counting in each quadrant is gross way of accessing fields. Complete visual fields done after the patient becomes stable.

PUPILLARY EXAMINATION:

Pupillary size, shape, irregularities of margin, directs and indirect light reflex is noted.

'SWINGING FLASH" test may demonstrate abnormal pupillary reaction. Neutral density filter may detect subtle RAPD. Anisocoria if any should be noted.

COLOUR VISION:

This is Useful in assessing optic nerve function especiallyin traumatic optic neuropathy. Ischihara's chart may be used. A red object such as top of a mydriatic bottle can be used to assess color desaturation.

EXTRAOCULAR MOTILITY:

Extra ocular motility abnormality is most likely to occur with orbital injuries where muscle entrapment or injury, orbital hemorrhage or cranial nerve injuries result in decreased motility.

Testing of ocular motility should be performed, but it may be complicated in periorbital edema, pain with examination or poor patient co-operation. Forced duction test can differentiate between neuropathy or muscle injury and muscle entrapment.

ANTERIOR SEGMENT EVALUATION:

First the orbit and surrounding Periorbital tissues are evaluated for laceration, ecchymosis, edema, lid abnormalities, enophthalmos, obvious bony deformities,

PALPATION:

In fracture orbital wall, irregularity of orbital rim, infraorbital hypoesthesia, crepitus and obvious foreign bodies if any should be noted.

EYELIDS:

The eye lid should be examined for small lacerations pointing towards occult penetrating injuries or lid margin lacerations which if left unrepaired could result in lid position abnormalities.

Partial thickness lacerations are meticulously repaired with interrupted or horizontal mattress suture. Full thickness lid injuries are sutured in layers with slight eversion of edges to prevent eye lid notch with 6-0silk.

CONJUNCTIVA:

The conjunctival surface should be inspected for evidence of ocular laceration or rupture with uveal prolapse or visible scleral defects. Large areas of dense bloody chemosis increase the likelihood of underlying ocular rupture.

Conjunctival hemorrhage may be due to fracture of the orbital wall or the base of skull. In this case, posterior limit of the hemorrhage cannot be made out while anteriorly it may not reach the limbus and the color is purplish. The blood gets absorbed from one to three weeks without treatment.

Scleral entrance wounds are more likely to be associated with ocular perforation in foreign body injuries.

CORNEA:

The corneal epithelium should be evaluated for the presence of staining defects; superficial foreign bodies are safely removed at slit lamp.

Deep stromal foreign bodies are removed in theatre under microscope.

Foreign bodies which traverse the cornea and extend into the anterior chamber should be treated as full thickness corneal wound and repaired in operating room.

Corneal Edema:

This is seen after severe concussion injuries due to temporary disturbance of the cells of corneal endothelium, their permeability being altered, so that aqueous gets free access to the corneal tissues. Mostly it clears up without specific treatment.

Corneal abrasions:

Superficial abrasions are commonly found in many cases .most of them are small and heal easily by itself.

Lacerations of the Cornea:

Any corneal laceration should be closely examined to determine depth and stability, using slit lamp inspection and seidel testing.

Corneal perforations can be non perforating corneal lacerations or full thickness corneal laceration.

Folds in Corneal tissues:

Folds in Bowman's membrane may be due to force, which directly impinges on the cornea (Lattice like opacities of Casper 1903). Folds in the descemet's membrane are much more common, producing a deep striate opacity. They may occur without any superficial abrasions in cornea. Pigmentary deposits on the posterior surface of the cornea are occasionally seen after a concussion. They are derived from the iris and are usually scattered diffusely over a wide area.

Blood staining of the cornea:

It results from a contusion injury that is associated with a massive hyphema and raised intraocular pressure. The damaged corneal endothelium permits erythrocyte fragments to enter the corneal stroma.

The affected area is stained at first a rusty brown or a greenish black, which gradually changes to greenish yellow or gray. It may take the form of a central ring (Manschot 1947) or disc (Schousboc and Morard 1935). The resultant blood staining clears spontaneously but it may require a considerable amount of time. The central cornea clears last.

Surgical intervention to remove blood from the anterior chamber and reduce IOP is indicated on earliest sign of corneal blood staining. Earlier intervention is done in patients with compromised endothelial function..

Corneo scleral laceration:

It is a rare event that occurs only on the impact of severe force directly upon eye. The sclera usually bursts at its weakest point near the cornea scleral margin. The presence of old cicatrix may increase the tendency.

Small ruptures near the limbus are associated with a prolapsed of the iris, while in a case of large rupture the lens, vitreous and varying proportion of uveal tissue may be extruded. In cases of corneoscleral laceration with iris prolapse, excision of the prolapsed iris tissue and accurate suturing of the corneal wound results in good healing.

Infectious microbial keratitis:

Ocular trauma is a frequent precipitating cause of infective microbial keratitis. The treatment is according to history, clinical findings and microscopic examination of corneal smear.

ANTERIOR CHAMBER:

The anterior chamber depth should be evaluated by slit lamp. There can be shallow anterior chamber due to corneal full thickness defect or anterior lens subluxation. An increase in depth of anterior chamber is seen in posterior dislocation of lens or depth is irregular in subluxation of crystalline lens.

Presence of flare cells, whether inflammatory, pigmented or RBCs should be noted. Any foreign body should be described. In a patient who has intact globe, gonioscopy should be performed looking for an IOFB in the angle.

Blunt trauma can produce traumatic iritis with photophobia and eye pain resulting from ciliary spasm. Slit lamp examination may show perilimbal injection with cells and flare.

TRAUMATIC HYPHAEMA:

Hyphema is graded by the amount of blood in AC.

Grade 1: less than ¼ of visible volume of AC.

Grade 2: one quarter to half of visible volume of AC.

Grade 3: one half to ¾ of visible volume of AC.

Grade 4: complete filling of visible AC.

Eight-ball hyphema refers to completely filled anterior chamber with black colored clots .gonioscopy is contra indicated since it may cause re-bleeding.

IOP should be monitored regularly.

Medical treatment:

Miotics, cycloplegics, fibrinolytics, estrogen and corticosteroids are useful.

Surgical intervention:

>50 mm Hg for 5 days or >35 mm Hg for 7 days to avoid optic nerve damage is the criteria.

Paracentesis and anterior chamber wash, clot expression and limbal delivery, automated hyphemectomy using cutting /aspiration instruments are various options. When pupillary block glaucoma in large hyphema extending into posterior chamber, occasionally peripheral iridectomy is also done.

ANGLE RECESSION:

A tear in the ciliary body itself results in angle recession. Angle recession is suspected at gonioscopy after 6 weeks by comparing the injured with uninjured eye in relation to peripheral depth at the limbus. Gonioscopy

reveal unevenness in width of ciliary body band or band greater than trabacular meshwork in width. If more than 180 degree is involved there is 10% chance of developing glaucoma.

Angle Recession Grade:

Grade I: - Ciliary body band appear darker and wider, scleral spur appear whiter than the fellow eye owing to tearing of the uveal meshwork.

Grade II: - (Moderate angle tears) angle is deeper than the fellow eye owing to tears in the face of the ciliary body.

Grade III: - (Deep angle tears) a deep furrow extends into the ciliary body, the apex of the fissure cannot be identified gonioscopically. Deep tears can be visualized by using anterior segment ultrasound bio-microscope.

Angle recession glaucoma was found to be the second most common mechanism of unilateral glaucoma. Glaucoma may develop months or years after injury. Eyes with less than 180 degrees of recession are unlikely to develop late glaucoma.

Patients with angle recession without glaucoma need to be followed annually for development of glaucoma. Tear in the trabacular meshwork seen in gonioscopy as torn iris processes, prominent scleral spur and circumferential tears in the surface of trabacular meshwork. They may heal by fibrosis contributing to glaucoma.

CYCLODIALYSIS CLEFT:

Cyclodialysis cleft is separation of ciliary body attachment to scleral spur. Gonioscopy reveal white area below the scleral spur. Ultrasound biomicroscopy (UBM) may be helpful.

IRIS:

Iris should be evaluated closely for injuries .Iris tear may be visible within stroma or at the sphincter or gross separation of peripheral iris from the eye wall may be seen indicating Iridodialysis or cyclodialysis.

Iris stroma should be evaluated for transilluminating defects, best seen in retro illumination, may show penetrating injury in RTA.

An incarcerated non prolapsing iris should rarely be removed. In peripheral iris incarceration and a well formed AC, acetyl choline in anterior chamber may constrict and release the iris.

If the incarceration is central, epinephrine dilates pupil and may release the iris.

Prolonged prolapsed iris leads to ischemia and subsequent necrosis. Repositioning should be made only when the exposition is for small time. Iris is abscissed if it is macerated, necrosed and exposed for more than 24 hours.

LENS:

The lens capsule should be inspected for areas of decreased lucency or obvious penetration. The lens body should be inspected for signs of penetrating injuries such as cataract, foreign body, or obvious disruption.

Pigment deposition on the anterior lens capsule (vossius ring), instability of the crystalline lens (phacodonesis) and displacement of crystalline lens with/without the presence of vitreous in anterior chamber(zonular dehiscence, luxation, or subluxation) may be present with blunt trauma involved in RTA. Pseudophakic patients may show instability, displacement or dislocation of implanted lens.

Posterior capsular status is importment in planning surgery, is by slit lamp or B-scan.

Subluxation and Dislocation of the Lens:

When more than 25 percent of the fibers are broken the lens is said to be subluxated, it may remain in the posterior fossa retained by its attachment to the vitreous.

A dislocated lens is a lens that is completely detached from its zonular and vitreous attachments.

A minimally subluxated lens may induce myopia caused by increased curvature of lens due to zonular disruption or astigmatism as it is decentered. If the lens dislocation is more and its edge is in the visual axis, monocular diplopia may develop.

Miotics are used to minimize diplopia and astigmatism produced by traumatic minimally subluxated lens. Mydriatics is used to enlarge Aphakic portion, so that Aphakic correction is given.

The lens may be mobile in the vitreous(Lens natans) and by changing the position of the patient from the prone to supine through the dilated pupil the lens travel from the vitreous into the anterior chamber(wandering lens) (Wolte 1945) but eventually organized membrane tend to anchor it. Very rarely it may be present subconjunctivaly.

Surgical approach:

Anterior segment methods:

Cataract with intact posterior capsule, no displacement, no vitreous in anterior chamber is removed by anterior approach.

A hard subluxated lens with extensive zonular damage if associated with extensive zonulardamageshouldbe extracted using intra capsular approach with scleral fixated lens or anterior chamber IOL implantation.

A minimally subluxated lens can be removed by phaco emulsification. Localisedzonular dialysis, either prior or during cataract surgery is an indication for capsular tension rings, when the zonular dialysis is less than 45 degree. If the zonular damage is more than this, involving 2 quadrants, then modified capsular tension ring is used.

Posterior segment method:

Pars plana lensectomy with vitrectomy is useful in significant posterior capsular disruption or posterior dislocation of lens and vitreous prolapsed into the anterior chamber and management of associated vitreous hemorrhage, intraocular foreign body, retinal detatchment, etc

As a rarity the lens may slip through a retinal tear into inter retinal space or it may lie between the sclera and ciliary body (sub scleral Luxation).

Concussion Cataract:

Mechanism:

When a sudden force strikes the eye, a wave of pressure thrusts the aqueous and iris, forcibly against the lens and pushes it backwards on the vitreous, on its rebound the lens curls itself against the iris. In the fluid contents of the globe the force is transmitted in all directions, so that capsule and its epithelium as well as lenticular substances itself are concussed.

Tears occur at the thin portion of the capsule covering the posterior pole of the lens. Sometimes they are covered by the iris, so rapidly sealed by fibrin and sub capsular epithelium, which secretes a new capsule. In these cases entrance of aqueous is stopped. So opacity in the lens may remain stationary. If the tear remains open the opacification progresses to involve the entire lens.

Rosette type cataract is usually seen in the posterior cortex, sometimes in the anterior cortex or both.

In surgical removal of traumatic cataract with intact posterior capsule, no displacement, no vitreous in anterior chamber or lens dislocation into AC, anterior approach via limbus is preferred.

A posterior pars plana approach for removing cataract with posterior capsule ruptured, lens dislocated or subluxated into vitreous posteriorly.

Lens induced intraocular inflammation may occur secondary to phaco anaphylactic uveitis.

Phacolytic glaucoma should be treated with corticosteroids and anti glaucoma medications and lensectomy. phacomorphic glaucoma can be treated by lens removal.

ANTERIOR VITREOUS:

The anterior vitreous should be examined for presence of IOFBs, particulate matter and or cells which could indicate infection.

POSTERIOR SEGMENT EVALUATION:

Posterior segment evaluation is must in all cases of trauma. It is done through direct and indirect ophthalmoscope.

In extensive ocular rupture, examination of posterior segment other than imaging should be deferred until wound is appropriatively treated.

Eyes with smaller, more stable wounds or blunt trauma without rupture may be safely dilated and examined to document the extent of injury and plan for surgical repair.

FUNDUS:

BERLIN'S EDEMA:

Commotio retinae, a cloudy swelling giving grey appearance involving temporal fundus, subsequent post traumatic macular changes like progressive RPE degeneration and macular hole should be looked for. Typically the edema is opposite the site of injury.

The edema may be localised confined to macula, posterior pole or peripapillary region. If the entire posterior pole is involved, a pseudo-cherry red spot may occur, mimicking central retinal artery occlusion. All this may subside in few weeks.

RUPTURE OF CHOROID:

Choroidal rupture may be direct, involving anterior retina at the site of impact running parallel with oraserrata or indirect rupture involving area opposite to the site of impact after a severe concussion injury.

Indirect ruptures are juxtapapillary in macular region, single and curvilinear. Choroidal rupture may be associated with vitreous hemorrhage. Further complications like choroidal neovascular membrane should be looked for.

RETINAL CHANGES:

Retinal breaks, holes retinal hemorrhage, retinal dialysis and retinal detachment should be looked for and managed accordingly. A traumatic retinal tear from blunt trauma is by transmission of external force to globe.

Avulsion of vitreous base from the peripheral retina is pathognomonic of ocular trauma, because the vitreous base is normally firmly attached to the retinal periphery and pars plana.

Retinal dialysis with or without retinal detachment is more common in supero temporal area. Traumatic retinal tears with or without retinal detachments can occur.

A giant retinal tear involves tears of more than one quadrant of retina.

Prevalence of retinal dialysis in traumatic retinal detachment according to a study is 84%,gaint retinal tears is 8%,horse shoe tear is 3% and round hole is5%.

Retinal tears with blunt trauma can be treated with photocoagulation or cryopexy. Retinal detachments are treated with scleral buckling, pneumatic retinopexy and vitrectomy.

TRAUMATIC OPTIC NEUROPATHY:

Pupil should be assessed for traumatic optic neuropathy for the presence of RAPD. Color vision and visual acuity are assessed.

Damage to optic nerve may be due to direct injury by fractured bone pressing over nerve or compression by hemorrhage. It may occur at intraocular, infraorbital, intracanalicular or chiasmal level.

Indirect optic nerve trauma refers to optic neuropathies that follow closed head trauma. The posterior indirect optic nerve traumas, typically damage the intracanalicular segment. The 10mm intra canalicular portion together with its meninges, the ophthalmic artery and sympathetic nerves are crowded tightly with in bony canal.

The damage could be due to shearing force on optic nerve at optic canal where it is tethered to dural sheath or transmission of shock waves through orbit.

CT scan of orbit and head is the initial study of choice. Decompression of optic canal is by craniotomy or through paranasal sinuses.

After ruling out fracture at optic canal, a course of high dose steroids, inj methyl prednisolone1000mg /day in divided doses for three days followed by tablet prednisolone 1 to 1.5 mg/kg for 11 days is given according to ONTT trial.

OPTIC NERVE AVULSION:

Optic nerve avulsion is rare where there is a striking cavity where the optic nerve head has retracted from dural sheath. Fundus shows a striking cavity in the area of optic nerve. The mechanism involved may be sudden

extreme rotation or anterior displacement of globe. Prognoses depend on whether the avulsion is complete or incomplete.

Purtcher's retinopathy:

Purtcher's retinopathy is a traumatic angiopathy, commonly caused by head and chest trauma with bilateral retinal signs including white ischemic infarcts with blot, preretinal and flame shaped hemorrhages.

OPEN GLOBE RUPTURE:

Scleral rupture can be occult,hidden under the conjunctiva, tenons capsule or recti muscle. Chemosis or Subconjunctival swelling suggest the presence of occult rupture. Slit lamp biomicroscopy and B-scan are useful. CT scan is useful in demonstrating rupture and radio opaque foreign body.

In many cases of penetrating injury it is necessary to repair a corneo - scleral laceration immediately. Following that some require pars plana vitrectomy. Even then the chance of endophthalmitis in open globe injury is 6.8%.

Traumatic Hypermetropia:

It is usually associated with paralysis of accommodation. May be temporary or permanent due to an injury to ciliary nerves or the ciliary muscles itself. Very rarely a traumatic hypermetropia is due to rising of the retina with organized material after a rupture of the choroid .

A posterior dislocation of the lens with an increased depth of anterior chamber will alter the refraction in the direction of hypermetropia.

Traumatic Myopia:

This is the commonest refractive change following a concussion to the globe. As a rule an increase in myopic refraction of the eye ranges from ID to 6D and disappear within a week or two.

Ciliary spasm accounts for majority of cases due to irritation of the muscle fibers themselves or irritation of the third nerve or paresis of cervical sympathetic chain. This account for 1D to 4D myopia. It disappears with atropine. There will be a spastic miosis.

Subluxation may produce about 5D or 6D myopia (Moller). Higher degrees may be produced by anterior dislocation of lens.

INTRAOCULAR PRESSURE TESTING:

IOP is tested using applanation tonometer. If any globe rupture is suspected, non contact tonometers are used.

IOP testing is deferred in open globe injuries. Patients with low IOP can have ciliary body dysfunction or occult ocular penetration.

IOP may be elevated in aqueous outflow obstruction, clogging of trabacular meshwork by hemorrhage, debris, foreign material or aqueous misdirection, Suprachoroidal hemorrhage or Pupillary block by dislocated lens.

ORBITAL FRACTURES:

Orbital wall blow out fracture is of two types:

1. Pure blow out fracture which does not involve orbital rim.

2. Impure blow out fracture involves the orbital rim and adjacent facial bones.

A blow out fracture is caused by increase in orbital pressure greater than 5cm, such as fist or tennis ball. The most common fracture is floor of orbit along with thin bone covering the infra orbital canal. Medial orbital wall may also be fractured. The lateral wall and roof is usually able to withstand such trauma.

Mid facial fractures are classified by LeFort classification.

LeFort 1 is not associated with orbital fractures.

LeFort 11 is associated with pyramidal maxillary fracture extending to medial floor and wall of orbit.

LeFort 111 fracture is extensive midfacial fracture involving lateral walls, floors and medial walls of orbit.

BLOW OUT ORBITAL FLOOR FRACTURE(#):

Orbital floor fractures are classified into direct and indirect.

Direct Forceful trauma on inferior rim result in zygomatic fractures involving zygomatic arch or its posterior articulation in addition to its lateral and inferior orbital rim. This three part zygomatic fracture is referred as **tripod fracture.**

1. Trap door type-large fragment of medial portion of floor of orbit is fractured but remains attached to laminar bar resemble a trap door.

2. Medial blow out-disruption of floor of orbit occurs between infraorbital nerve and laminar bar.

3. Lateral blow out-there is communition from the laminar bar to lateral wall.

SIGNS:

1. Periocular signs-Ecchymosis, edema,subcutaneous emphysema.

2. Infra orbital anesthesia due to involvement of infra orbital nerve.

3. Diplopia-due to

a. Hemorrhage and edema in orbit which makes septa connecting inferior rectus and inferior oblique to periorbita become taut.

b. Mechanical entrapment of inferior rectus or inferior oblique in the fracture.

c. Direct injury of extra ocular muscle associated with negative forced duction test.

1. Enophthalmos

2. Hyphema, angle recession, retinal dialysis.

X-RAY PARANASAL SINUS:

Classic tear drop sign seen.

Caused by prolapsed of orbital contents into the maxillary sinus.

Corresponding maxillary sinus is hazy.

Hemosinus is common.

CT SCAN:

Coronal view is useful in evaluating fracture and prolapsed orbital tissues.

Enophthalmos and diplopia develop mainly following indirect or blow out floor fractures. Orbit is decompressed by blowing out the thin bones of floor. Small orbital floor # is more likely to cause entrapment due to trapdoor effect. Large # causes enophthalmos.

When non-displaced, these fractures do not require fixation. Direct inferior rim fractures and tripod fractures rarely affect ocular motility.

Surgery is indicated in patients with

- diplopia in primary gaze with muscle entrapment
- large floor fractures with enophthalmos>2mm
- progressive numbness in infraorbital areas
- severe -hypophthalmos.

Though various approaches including trans-lid-infra orbital rim, sub ciliary, Caldwell-Luc, Trans conjunctival incision, periostium elevated, entrapped tissues freed, fracture stabilized with autologous /alloplastic material.

Timing of surgical intervention is easier in terms of scaring and fibrosis if performed within first 10 days of injury. But it is better to wait for a few days for some resolution of soft tissue swelling and hemorrhage. If acute intervention is not warranted wait for 6 weeks.

BLOW OUT FRACTURE OF MEDIAL WALL:

Most medial wall fractures are associated with floor fractures.

TYPES:

TYPES 1: pure medial wall fracture

TYPE 2: medial wall and floor fracture

TYPE 3: medial wall, floor and trimalar fracture

TYPE 4: medial, floor, maxillary nasoorbital and frontal bones

In Type 1-fracture of medial wall orbit is by assault. Other types are by RTA.

Visual disturbance common in type 1, 2, 3, but not in type 4.

Eye ball injury is more common in type2.

Diplopia and enophthalmos is more common intype2.

Displacement of orbital wall and prolapsed of orbital contents uncommon in type1, 2, and 4 but common in type 3.

SIGNS:

1. Periorbitalhematoma, sub cutaneous emphysema-after blowing nose

2. Defective abduction, adduction

CT SCAN:

To know the extent of damage.

When medial wall # is associated with orbital floor #, enophthalmos is more and surgical repair should be done as orbital floor #.

FRACTURE LATERAL ORBITAL WALL:

1. Itis associated with fracture of zygoma and malar complex.

2. This fracture is common in adults.

3. This fracture should be suspected in severe fascial injuries.

FRACTURE ROOF OF ORBIT:

This type of fracture is quite rare.

Associated with falling on sharp objects or blow to brow /fore head.

Signs:

1. Hematoma of upper eye lid and peri ocular ecchymosis.

2. Inferior or axial displacement of globe.

3. Transmission of CSF pulsations.

Roof # is treated conservatively with IV antibiotics, surgical intervention only in the presence of complications like fracture of optic canal or frontal bone displacement.

INTRA OCULAR FOREIGN BODIES:

Intraocular foreign bodies (IOFBs) are commonly encountered in ocular injuries. Up to 40% of eyes with open–globe injury will contain at least one IOFB.

Retained IOFB is a true emergency that can lead to severe vision loss due to endophthalmitis, retinal detachment, ciliary body dysfunction, ocular metallosis and even loss of eye since IOFB injuries are costly, both economically and personally.

Most of the eyes with retained IOFBs maintain good vision with appropriate treatment. Visual prognosis is best when IOFB is removed during the initial wound repair surgery.

MECHANISM OF INJURY:

Majority of IOFBs are small sharp projectiles produced from hammering metal or stone. Up to 90% of IOFBs are metallic and 55-80% of these are magnetic. Since they travel at high velocity they are sterile. They penetrate and cause ` ocular disruption. Motor vehicle accidents are important cause of IOFB. Other causes are assault, insect stings, explosion and use of machine tools.

Common IOFBs:

Iron,lead,copper,zinc,silver,gold,platinum,nickel,plastic,woodand glass. Majority of patients with IOFB are male.

Route of entry and location of foreign body:

IOFBs most commonly enter the eye through the cornea (65%). Other common locations for penetrating lacerations include the sclera (25%) and limbus (10%). The IOFB most frequently in the vitreous cavity (61%), but can

also be located within the anterior chamber (15%), retina (14%), and lens (8%) or sub retinal space (5%).

Investigating intra ocular foreign body:

The diagnosis of an IOFB begins with a thorough history and suspicion for its presence. History should include circumstance of trauma, the elapsed time since injury, the use of safety glasses, and any exposure to hammering, grinding, drilling, or an explosion.

History of trauma and ocular signs such as localized lenticular opacity, a self sealing corneal or scleral wound, mild intraocular pressure asymmetry, a minor change in shape of the pupil, or mild iris heterochromia, may be all that suggest the presence of an IOFB.

Important information to document at the initial examination includes baseline visual acuity, papillary reaction, intraocular pressure, external examination, slit lamp biomicroscopy, assessment of media clarity, extent and location of wound, iris color, lens status and presence of retinal tears and detachment.

The size, shape, location, number, type, magnetic properties, and entry path of foreign body should be fully described. IOFB composition and its magnetic properties are especially important since they influence prognosis and method of IOFB extraction.

Prognosis and complication:

When an intra ocular foreign body is established, prognosis is always guarded.

Complications

Siderosis:

This is a late occurring syndrome caused by retained foreign body. Initial effects are mechanical and contusive. It is uncommon to have infections as the heat sterilizes the metal.

Iron interacts with intraocular tissues by electrolytic dissociation causing gradual disappearance of foreign body and spread of ferric ions throughout the globe.

In Siderosis,ferrous pigment causes rusty coloration of cornea, iris or lens. The predilection of copper for deposition on limiting membranes is the presence of electric current. Copper interaction with intra ocular fluids and tissues causes ionization and its distribution on limiting membranes by electrostatic factors.

Later as chronic degeneration occurs it may lead to cataract, pigmentary degeneration of retinal pigment epithelium and Muller cells, retinal detachment or open angle glaucoma. This may take two weeks or two years to develop.ERG shows decrease in b-wave amplitude in metallosis bulbi.

Chalcosis:

Copper and its common alloy bronze and brass may induce Chalcosis. If the copper content is high more severe reaction occur including hypopyon and localized scleral abscesses.

Acute Chalcosis mimic pyogenic Endophthalmitis.

A K-F ring in descemets and a sunflower cataract in anterior capsule are manifestations of Chalcosis which resemble Wilson's disease.

Anterior chamber has retractile particles, iris becomes green and sluggish in reaction .Retina may have shining particles along vessels.

Sometimes retained foreign body may cause destruction of neurons and vision loss.

REMOVAL OF INTRAOCULAR FOREIGN BODY:

SUPERFICIAL FOREIGN BODIES:

Foreign bodies lodged in cornea and conjunctiva does not produce any serious damage.

A foreign body in bulbar conjunctiva is removed by cotton tipped applicator without anesthesia and in tarsal conjunctiva is removed after double eversion of upper eye lid.

Corneal foreign body can be removed by foreign body spud after topical anesthesia. Deeper ones removed under operating microscope. Glass particles

are most difficult ones to remove.Vegetable foreign bodies are associated with infection.

ANTERIOR SEGMENT FOREIGN BODIES:

Foreign body's entering and remaining in anterior segment of eye can be removed through wound of entry at primary surgical repair.

Depending on size and nature of foreign body larger incision at limbus can be made for foreign body at angle.

POSTERIOR SEGMENT FOREIGN BODIES:

Giant magnet, permanent hand magnet, hand electro magnet and Bronson-magnion instrument were previously used for foreign body removal.

Non-magnetic foreign bodies:

Surgical maneuvers are difficult. Usually direct Trans scleral extraction is done to minimize trauma sclerotomy must directly over the foreign body.

If the foreign body lays intra vitreally then use forceps or other devices to grasp the foreign body through parsplana route.

Posterior segment foreign bodies obscured by opaque media, too large for pars plana route require pars plana vitrectomy to ensure a minimally traumatic extraction.

When a foreign body is present over retina near macula, per fluro carbon liquid is used to float the foreign body before removal.

Intra orbital foreign bodies:

It may remain without symptoms and signs. It may produce granuloma, orbital cellulites, periostitis, orbital abscesses, osteomyelitis, and draining fistulas.

If it strikes orbital apex, blind, anesthetic and immobile globe result.

INVESTIGATIONS:

PLAIN FILMS:

Plain X-rays are valuable screening tool for the evaluation of orbital fractures and intraocular and intra orbital foreign bodies. With the advent of CT and MRI, the plain film is used often for purposes other than rapid screen.

IMAGING:

ULTRASOUND:

Ultra sound means high frequency sound waves. These are above the audible range.

In ophthalmology a-scan and b-scan are used for various diagnostic purposes.

Diagnostic ultrasound is significantly different in all three properties from the previous two categories.

1. It does not generate heat in the ocular tissues.

2. It does not use high power sound energy.

3. Its frequency is much higher in the range of megahertz.

Higher frequency of 10 MHZ is commonly used in ophthalmic ultrasound.

FOREIGN BODIES IN ULTRASONOGRAM:

Foreign bodies in the eye present some unique artificial echoes. In fact, these artifacts are quite useful in making an evaluation of what the material might be.

The artifacts created by foreign bodies may be grouped into two categories-those with:

- Extra echoes generated

- Lack of echoes posterior.

It can detect both radiolucent and radio dense IOFBs in a study it has identified all foreign bodies including a cilium. It can show the precise location of IOFB especially when it is adjacent the sclera.

It can also demonstrate ocular abnormalities including retinal and choroidal detachments, vitreous hemorrhage and exit wounds.

Higher frequency ultra sound is useful for more accurate description of IOFBs shape size or relative position. This is used in anterior chamber IOFB.

It can differentiate a small metallic foreign body from air bubble introduced by trauma.

Though ultra sound can be done over lid in open globe injuries, it should be performed cautiously.

Ultrasound is usually not done in open globe injuries to avoid prolapsing of ocular contents. But it can be done intra operatively, after the entrance wound is closed.

COMPUTERISED TOMOGRAPHY (CT SCAN):

CT scan is readily available in most hospitals and is ideal for imaging bony orbital structures. When used with 1.5-3.0mm sections in the axial and coronal planes, it is superior to ultrasound in determining the size and location if IOFBs.

However, the intraocular structures are less well imaged than with ultrasound. The scanning technique does not require contact with the ocular surface and so may be used in patients with open globe injuries.

CT scan can suggest an intra ocular foreign body's density. But CT scan may not detect fragments that are smaller than 0.7mm in dimension, composed of wood or lying undetected in sclera.

CT scan findings indicative of open globe injury include the presence of intraocular air or foreign body, deformity of eye wall, and the presence of intraocular hemorrhage.

CT scan artifacts may be minimized by appropriate technique if the radiologist is notified of a possible foreign body near the posterior eye wall, increasing the accuracy of intraocular vs. extra ocular localization.

CT scanning is less expensive than MRI and, unlike MRI, may be safely used to image metallic foreign bodies. CT scan is generally contraindicated in pregnant patients.

MAGNETIC RESONANCE IMAGING (MRI):

Magnetic resonance imaging is better able to image soft tissues than is CT scan. It may be useful in the evaluation of patients with known non magnetic IOFBs and used in pregnant patients. It is often the only test capable of detecting small plastic or wood IOFB.

But MRI is more expensive and less available. It produces motion artifacts. It cannot be used in patients with pacemakers or implanted metallic hardware. MRI is not a screening tool and used after CT scan has excluded metallic foreign body.

It employs strong magnetic forces and can produce movement of magnetic foreign body and cause further ocular and neurological damage.

In cases of acute trauma, CT scan is preferred to MRI because of its lower cost ,more rapid results, and ability to image metallic foreign bodies

ULTRASOUND BIOMICROSCOPY (UBM):

UBM is high frequency ultrasound (35-50MHZ) with axial resolution as small as30 microns in imaging anterior segment of eye. The penetration is poor.

Anterior segment trauma may be associated with hyphema, where it is difficult to visualize iris and lens.UBM is useful to study the lens position, status of iris, ciliary body and configuration of angle. Angle recession and cyclodialysis cleft can also be evaluated.

ELECTROPHYSIOLOGY:

The electroretinogram (ERG) can document positive retinal function in severely injured eye. The visual evoked response (VER) can estimate visual potential. This may be helpful for physician in early post trauma repair time

when decision making is centered on enucleation /evisceration Vs secondary reconstruction.

A non–recordable bright flash ERG in the presence of a massive vitreous hemorrhage may not necessarily indicate permanent visual loss.

AIM OF THE STUDY

To study the magnitude and effects of ocular injuries due to road traffic accidents (RTA) and various factors associated with it.

MATERIALS AND METHODS:

This is a study carried out in the patients who suffered RTA and treated as outpatients and inpatients in Stanley medical college and hospital during a period of 2 Years patients were taken for study from October 2009 to September 2011 and follow up for each case is a minimum 6 months. This is a randomly conducted study and demographic data and details of injuries obtained.

The cases that came to the ophthalmology department were taken for the study.175 cases that came to outpatients were included in random fashion and entered in a pre structured profoma. The sample also includes those who were grossly injured and with limitation of vision.

Information regarding time, location, type and mechanism of injury use of spectacles, car safety belts and helmets were obtained.

Vision, eye findings, diagnostic tests, medical and surgical treatment were recorded. Tests were all done free of cost at Stanley hospital.

Whenever necessary, X-rays, ultrasonography, CT-scan which the patients had were utilized for the study.

A sample of the profoma used for the study is enclosed later..

INCLUSION CRITERIA:

1. Patients who suffered road traffic accidents within 7days.
2. Age group: >10 to <60 yrs.
3. Patients with no previous head injuries.
4. Minimum follow-up for 6 months.

EXCLUSION CRITERIA:

1. Head injuries and ocular trauma by modes other than RTA.
2. Age<1oyrs and>60 yrs.
3. Previous history of head and ocular injuries.
4. Poor follow up < than 6 months.

OBSERVATION AND DISCUSSION

The observations made during the study are as follows.It is compared with various studies of trauma and with a few studies of ocular trauma and road traffic accidents.

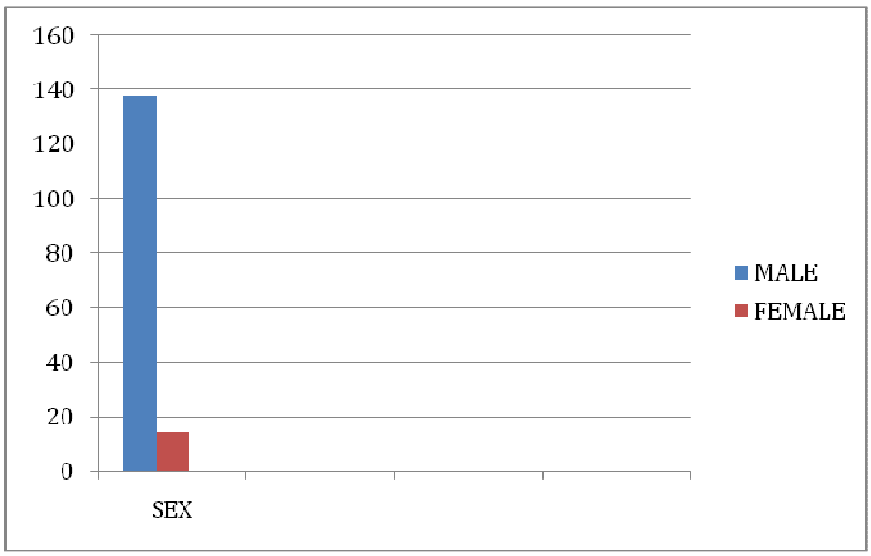
Out of 175 patients,23 patients did not report back for the 6 month follow up due to various reasons and 152 patients were finally included in the study.The obsevations made are as follows.

OCULAR INJURY DUE TO RTA IN MALES AND FEMALES:

Table 1:

Cause of injury	Male	Percentage	Female	Percentage
RTA	138	90.78	14	9.2

Figure 1:



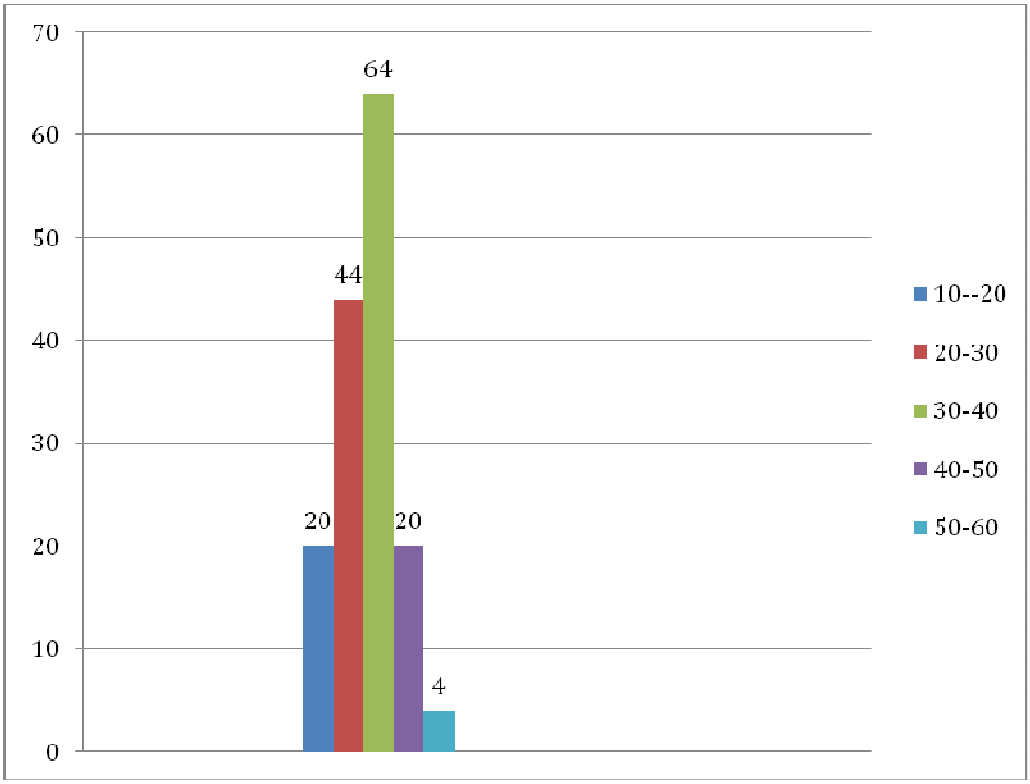
There were about 138 males and 14 females in the study. This clearly shows male preponderance in road traffic accidents, as shown in other studies also. This may be due to the fact that males are more exposed to the highway traffic when compared to females. In various studies it varies from 10:1 to 7:1. Here in our study about 90% of the study group was males, i.e. 10:1.In a study **by KAMATH.S.J** in2007[27] the ratio is 10:1 as in this study.

AGE AND OCULAR INJURY:

Table 2:

age	Number of patients	Percentage
10-20	20	13.1
20-30	44	28.9
30-40	64	42.1
40-50	20	13.1
50-60	4	2.6

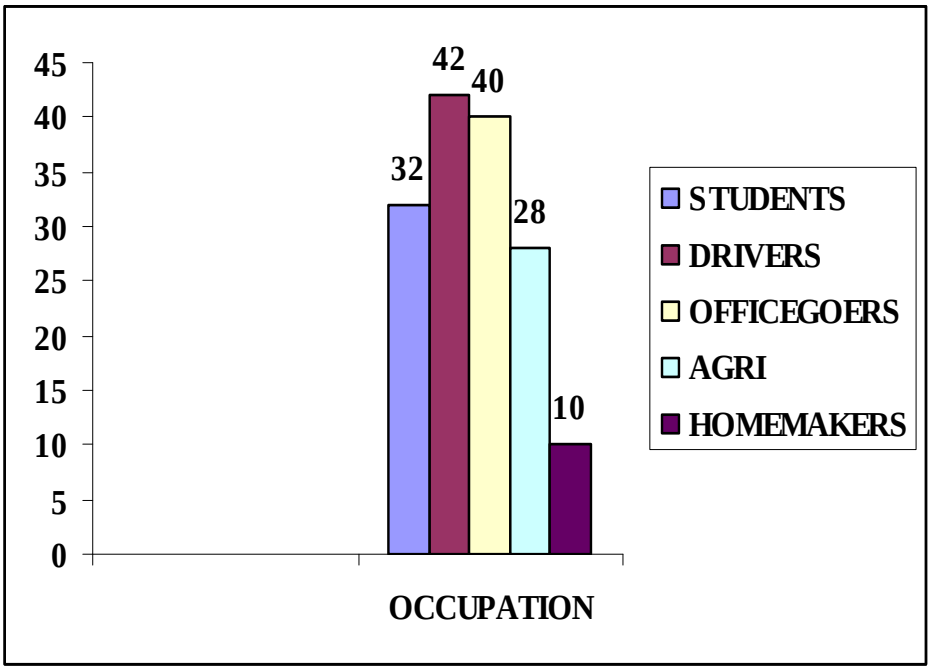
Figure 2:



About 20 (13%) people were of age between10-20, mainly teen agers. Majority of those involved were from 20-30(28.9%) and from 30-40 yrs (42%).A very few were above 50 yrs. At Tripoli study, mean age of patients was 32.5% and they included all age groups in the study[24]. In various recent studies the mean age is less than 40 years. In urban study at Delhi the mean age was28.21.[23]

OCCUPATION AND RTA:

Figure 3:



As already seen more young people of age 20 are involved in road traffic accidents and hence about 32 people (21.05%) were students.

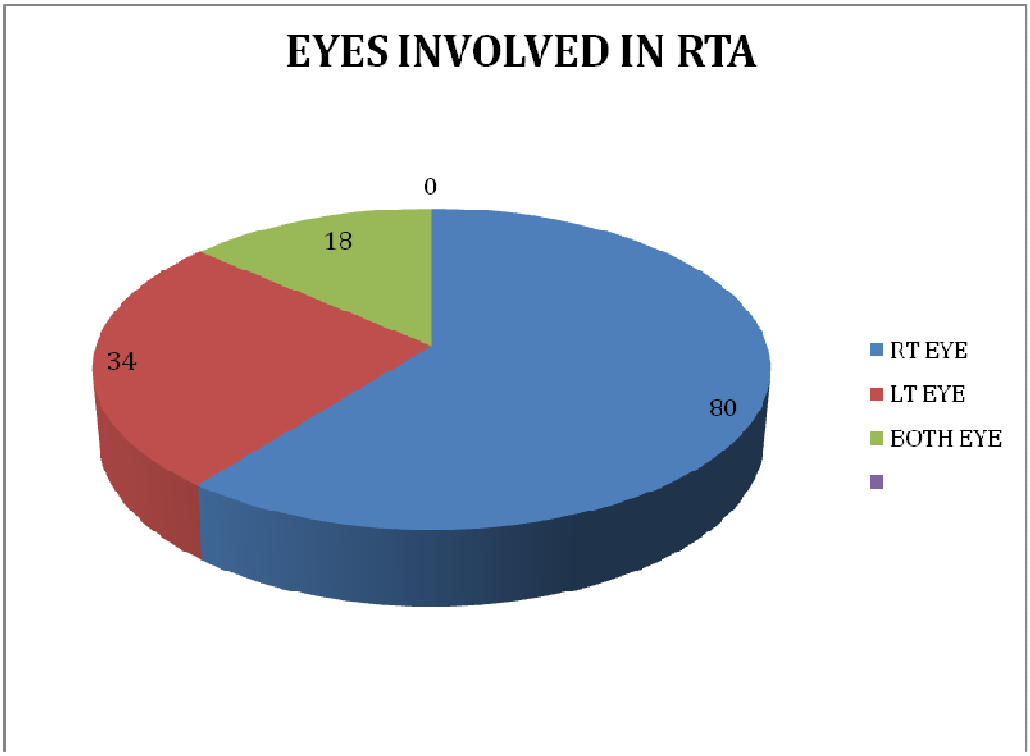
Many people involved in trauma were bread winners, mostly drivers and office goers. Among them were 42 drivers (27.63%) and 40 office goers (26.3%).

28 people (18.4%) were agricultural workers mainly from areas around Chennai and some were referred from other hospitals for their other ailments. A few of them were women (10).

Those who were riding 2 wheelers were mainly students, drivers and office goers. This indirectly shows more increasing number of 2 wheelers and their use by younger population. Those driving 4 wheelers were persons involved in professional driving. Privately owned cars were less involved in accidents.

EYES INVOLVED IN RTA:

Figure 4:



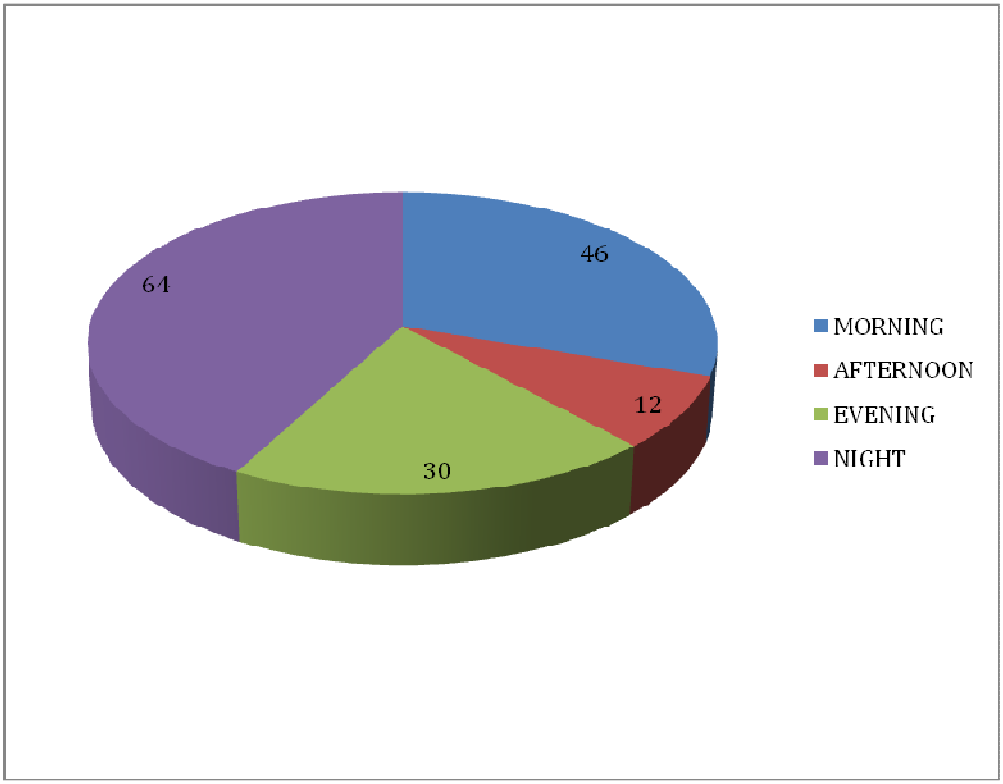
Eyes involved were mainly right about 80(52.6%). About 34(22.3%) were left eyes. Both eyes were involved in 18(11.8%). the Tripoli study on RTA shows that 42% involvement of right eye ,37.7% involvement of left eye and 20.3% of both eyes.

Time of injury: The following table shows accidents during different time of a day.

Table 3:

Time of accident	Number of patients	Percentage
Morning	46	30.2
Afternoon	12	7.8
Evening	30	19.7
Night	64	42.1

Figure 5:



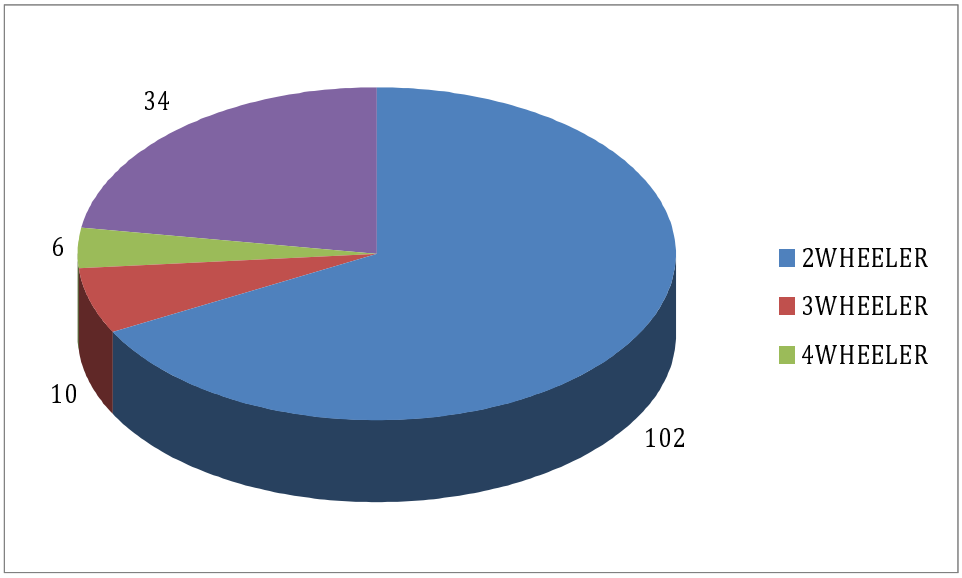
Time of injury affect trauma in many ways. The above chart clearly shows that trauma especially involving eye occur mostly at night. Almost more than one third occurred at night (42.1%).

The reason could be poor light, poor roads, traffic pattern and consumption of alcohol especially during night after a day's tiring work may be the reasons. During afternoon, when the movement of vehicles is less, accidents are less.

RTA-What vehicle was involved:

The different types of vehicles involved by patients in RTA have been described in the following diagram.

Figure 6:



Among those involved in RTA, majority of them were driving 2 wheelers 102 (67%).people in Autos were involved in 6% of cases.34 (22%)

cases were pedestrians hit by 2 or 3 wheelers. Just 3% of the patients were riding 4 wheelers.

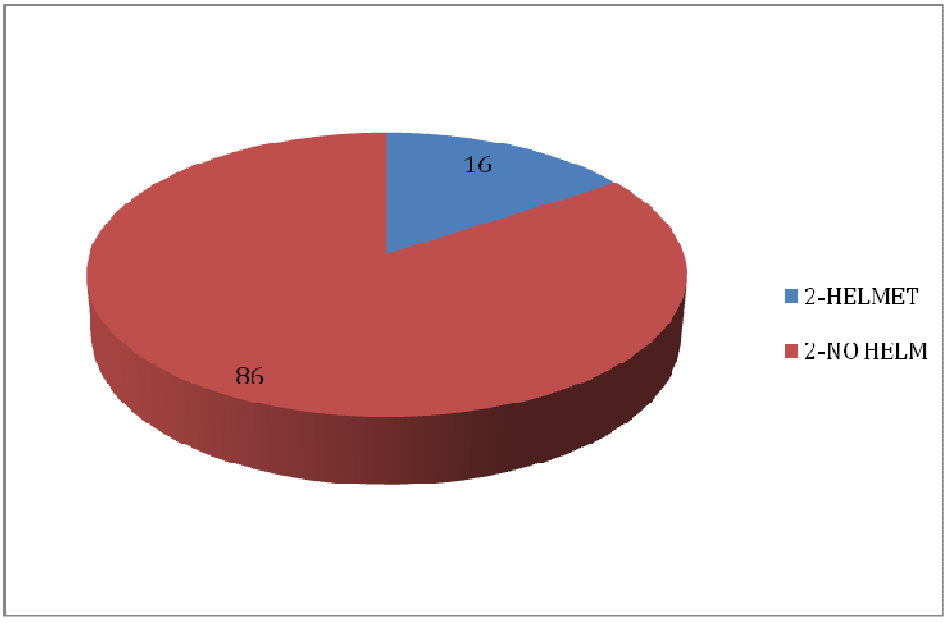
The vehicles which caused the accidents were mainly Lorries, motor bikes and cars. Autos and vans were involved. Many of these vehicles did not have proper head lights and rear lights, which were also the reason for accidents at night.

Some accidents occurred due to the carelessness of the drivers. Stray dogs were responsible for many accidents in and out of the city.

The study of eye injuries in Tripoli recorded that road traffic accidents were mainly due to 4 wheelers.[24]

RTA – Two Wheeler Accident and Helmet Use:

Figure 7:



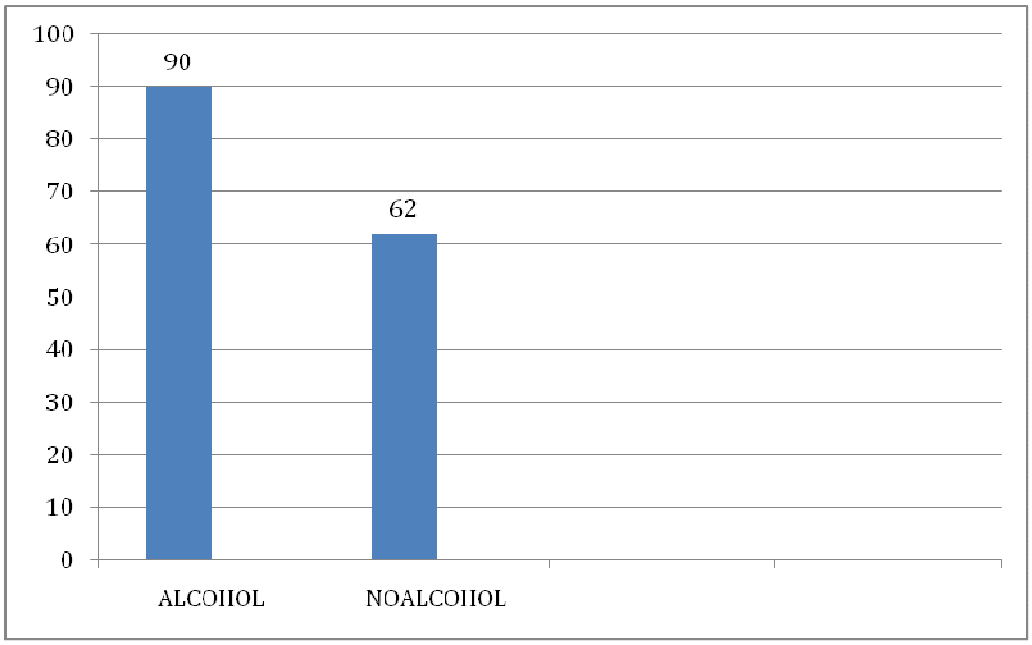
The above chart shows that among 152 patients, 102 used 2 wheelers. Of them, 86 patients did not use helmet. But only 16 of them used helmet which is only 15%. This attributes to the severe head and eye injury sustained by them. The use of helmet is seen as disturbance and a cause of more sweating among 2 wheeler drivers.

Influence of Alcohol on RTA:

Table 4:

RTA - Influence of Alcohol	Yes	No
	90	62

Figure 8:



Out of 152 patients, 90 patients (59.3%) were under the influence of alcohol while driving vehicles or walking along the road irrespective of age group .62 patients (40.7%) had not taken alcohol during the accident. This has a major influence on the occurrence of the accident. Those accidents which occurred during night were mainly under the influence of alcohol. Alcohol intake is assessed by history, breath analyzer test and the condition of the patient during earlier presentation.

Common ocular signs in Road Traffic accident:

Anterior segment findings:

The following table shows the findings in the trauma patient's anterior segment involved in the study. Various findings are as follows.

Table 5:

Road Traffic Accident	Number of injuries	Percentage
Periorbital edema	133	87.5
Periorbital ecchymosis	72	47.36
Orbital fracture	13	8.5
Eyelid edema	133	87.5
Eyelid laceration	21	13.8
Conjunctival chemosis	46	30.2
Subconjunctival hemorrhage	141	92.76
Corneal edema	6	3.9
Corneal abrasion	14	9.2
Corneal laceration	6	3.9
Traumatic iridocyclitis	21	13.8
Iris prolapse	4	2.6
Iridodialysis	5	3.2
pupillary sphincter tear	13	8.5
Traumatic mydriasis	10	6.5
Traumatic miosis	4	2.6
Anterior chamber hyphema	5	3.2
Traumatic cataract	6	3.9
Subluxated lens	8	5.2

EYE LID:

Table 6:

Eyelid tear IN RTA	RTA STUDY	URBAN STUDY*
PERCENTAGE	13.8	8.2%

* **EPIDEMIOLOGICAL STUDY OF OCULAR TRAUMA IN URBAN SLUM POPULATION IN DELHI BY S.VATS AIIMS-2007.**[23]

A common sign in RTA associated trauma is eye lid edema along with peri-orbital edema (87.5%). Tripoli study has recorded Periorbital edema of 35.5%.

Eye lid laceration was found in 21 patients (13.8%). 8 mild partial thickness tears were present. Those 13 patients with severe lid tears required suturing. Mechanical ptosis noted in 23 patients and1 case of aponeurotic ptosis noted.

CONJUNCTIVA:

Subconjunctival hemorrhage (SCH) is the most common eye sign in conjunctiva.

Table 7:

SCH IN RTA	RTA STUDY	HEAD INJURYSTUDY*
PERCENTAGE	92.76%	19%

* **OCULAR MANIFESTATION OF HEAD INJURY BY KULKARNI-EYE, 2004**

From the above tables it is evident that sub conjunctival hemorrhage (92.7%) is the most common eye sign associated with RTA in eye. Dense Subconjunctival hemorrhage should be looked carefully with slit lamp since it may mask a scleral laceration.

CORNEA:

6 patients had corneal edema. 3 patients had severe corneal lacerations and were repaired at OT. 3 patients had mild lacerations. Corneal abrasion of varying degree is a common sign in cornea. Some healed completely without scar, while some healed with corneal scar.

CORNEAL LACERATIONS	RTA STUDY	HEAD INJURY STUDY*
PERCENTAGE	3.9%	1%

ANTETIOR CHAMBER (AC):

Traumatic iridocyclitis was found in 21 patients.5 patients had developed hyphema. Out of 5 patients, 2 were of grade 4 completely filing the AC and 1of them were of grade 2.2 patients were of grade 1 occupying less than quarter volume of AC.

HYPHAEMA:

HYPHAEMA	RTA STUDY	URBAN STUDY*	HEAD INJURY STUDY**
PERCENTAGE	3.2%	0.6%	0.5%

* **EPIDEMIOLOGICAL STUDY OF OCULAR TRAUMA IN URBAN SLUM POPULATION IN DELHI BY S.VATS AIIMS-2007.**[23]

** **OCULAR MANIFESTATION OF HEAD INJURY BY KULKARNI-EYE, 2004**[25]

PUPILLARY INVOLVEMENT:

PUPILLARY INVOLVEMENT	RTA STUDY	HEAD INJURY STUDY**
PERCENTAGE	8.5	6.5

* **OCULAR MANIFESTATION OF HEAD INJURY BY KULKARNI-EYE, 2004**[25]

Pupillary involvement is more in RTA when compared to ocular injury associated with head injury due to other causes too.

IRIS:

4 patients with corneal and scleral tear had iris prolapse. Pupillary margin is inspected for sphincter tear.13 patients had pupillary sphincter tear.10 patients had traumatic mydriasis and 4 patients had traumatic miosis.6 patients showed relative afferent pupillary defect in the side of affected eye.

LENS:

6 of the 152 patients had traumatic cataract.4 of them had the posterior capsule was intact found using B-scan and in 2 cases vitreous was disturbed and vitreous found in AC. 8 cases had subluxated lens.

TRAUMATIC CATARACT	RTA STUDY	URBAN STUDY**
PERCENTAGE	3.9	19.6

** **EPIDEMIOLOGICAL STUDY OF OCULAR TRAUMA IN URBAN SLUM POPULATION IN DELHI BY S.VATS AIIMS-2007.** Traumatic cataract is only 3.95% in RTA when compared to urban slum study.[23]

INTRAOCULAR AND INTRA ORBITAL FOREIGN BODY:

Out of 152 patients, 6 patients were found to have intra ocular foreign body in this study 3.9% had intra ocular foreign body, while according to other studies it could be up to 40% with penetrating injuries. Intra orbital foreign bodies were found in 4 patients only accounting to 2.6% of total cases.

Posterior segment findings:

The table shows different findings of posterior segment of the eyes following injury.

Table 8:

Posterior segment findings	No of patients	percentage
Vitreous hemorrhage	6	3.9
Choroidal rupture	2	1.3
Retinal edema	4	2.6
Retinal break	6	3.9
Retinal detachment	4	2.6

In the posterior segment findings the most common finding was that of vitreous hemorrhage and retinal tears. 4 patients had retinal edema. Choroidal rupture was found in 2 patients. One of them developed choroidal

neovascularisation after 6 months 6 patients had retinal tears and 4 developed retinal detachments.

6 cases of vitreous hemorrhage noted. Various types of retinal hemorrhages were also found in various patients. Multiple Pre retinal hemorrhages were associated with vitreous hemorrhage.

VITREOUS HEMORRHAGE:

	RTA STUDY	**HEAD INJURY STUDY***
VITREOUS HEMORRHAGE	3.9%	0.5%

* **OCULAR MANIFESTATIONSOF HEAD INJURY BY DR KULKARNI A.R.EYE-2004**[25]

This shows that vitreous hemorrhage is more in RTA than with other ocular trauma.

RETINAL DETATCHMENT:

According to a study **ROLE OF USG IN OCULAR TRAUMA-IJRI-2001**[26]-vitreous hemorrhage is the most common finding in posterior segment as in this study.

	RTA STUDY	URBAN STUDY*
RETINAL DETATCHMENT	2.6%	1.3%

* **EPIDEMIOLOGICAL STUDY OF OCULAR TRAUMA IN URBAN SLUM POPULATION IN DELHI BY S.VATS AIIMS-2007.**[23]This shows that retinal detachment incidence is higher in trauma due to RTA.

TRAUMATIC OPTIC NEUROPATHY:

Table 9:

TRAUMATIC OPTIC NEUROPATHY	RTA STUDY	URBAN STUDY *
PERCENTAGE	3.9%(6)	**0.6%**

There were 6 cases of RAPD with severe limitation of vision. Those patients did not have pallor of disc immediately following injury. But they **developed pallor later.**

According to this study, traumatic optic neuropathy occur in 3.9%, while it is 0.6% in ***epidemiological study of ocular trauma in an urban**

slum population in Delhi by S.VATS-AIIMS, 2007.[23]This shows that optic neuropathy is more with RTA than with general ocular trauma.

Methyl prednisolone 500 mg IV bd for 3 days followed by T.prednisolone 1mg/kg for 11 days is usually given medical therapy.

In extended regimen, nowadays, injection methyl prednisolone is given for 5 days followed by T.prednisolone for 11 days. 4 patients who had PL + and one with PR defective did not improve in vision, while 1 of them improved from 2/60 to 6/60 and another improved from 3/60 to 6/60 with high dose of systemic steroids given immediately within 24 hours of trauma.

* Indirect optic nerve injury involvement in 2 wheeler riders in north east India shows that methyl prednisolone does not improve the outcome of indirect traumatic optic neuropathy.in our study 2 cases improved in vision while 4 cases did not improve.[11]

* **Indirect optic nerve injury in 2 wheeler riders in north east India IJO, 2008, BY HARSHA BHATACHARJEE.**[11]

VARIOUS TYPES OF ORBITAL WALL FRACTURES IN RTA:

The various orbital wall fractures found in the study are enumerated below.

Table 10:

TYPE OF FRACTURES	NO OF PATIENTS
SUPERIOR WALL	1
MEDIAL WALL	3
LATERAL WALL	5
INFERIOR WALL	4

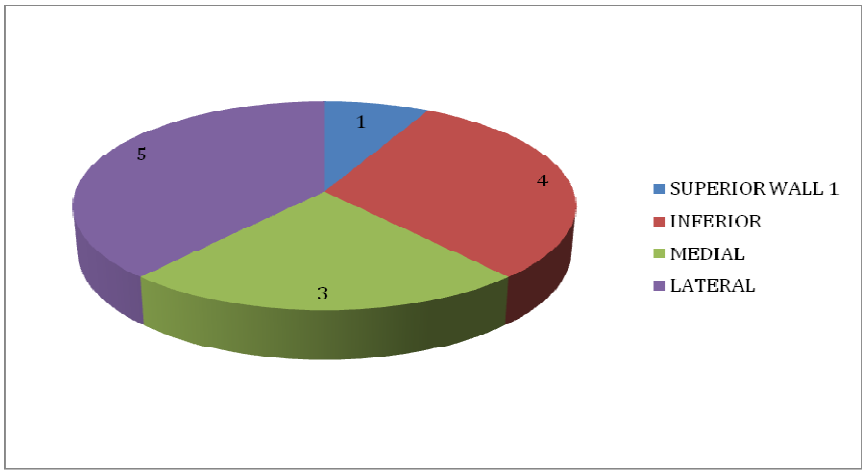
Out of 152 patients, 13(8%) patients sustained orbital wall fractures. Lateral wall fracture being the most common of all. Multiple fractures involving facial bones occurred in 5 patients.

Those with medial wall fractures were associated with crepitus. Inferior wall fractures were associated with maxillary wall fractures and hemosinus of maxilla.

One patient had superior wall fracture with head injury and was treated for it.

According to various studies study the percentage of ocular rim fractures was as little as 1.1%, to 10 % whereas in this study at Chennai it is 8% Here mainly 2 wheelers were involved leading to more severe type of injuries and lateral wall and floor fractures are more common. Lateral wall injuries may be more common due to the fact that that 2 wheelers when skid can lead to more of lateral facial injuries. Helmet wearing has an effect on the fracture of orbital wall. Those wearing helmets were less prone for fracture.

Figure 9:



Especially due to fall on the sides from 2 wheelers without wearing helmets produced fracture of lateral wall, while in all other ways in which eye injuries occur like blunt injury by fist, medial wall fractures are common.

RTA study &* orbital fracture study[27]

Floor	30%	19.6%
Lateral wall	38%	13.7%
Roof	7%	2.9%

***A study of orbital fractures in a tertiary health care centre by **dr kamath** .s .j 2007.[27]The study by **Battacharjee** on indirect optic nerve injury in 2 wheeler riders conclude that supero temporal orbital rim # are more common in 2 wheelers about 25% of the total cranio-orbital injuries. In this study lateral wall # is about 38% .2 cases of medial wall # had adduction restriction.2 cases of floor # had elevation restriction. None of the cases included in the study had endophthalmitis and panophthalmitis.

RESULTS

1. Out of 152 patients, 138 were males and14 were females which is about 10:1 ratio.

2. Regarding age distribution, the maximum numbers of patients were in the age group between 20 to 30 and 30 to 40, about 108 which accounts for 42.1%.

3. Occupation of the patients shows that the risky group in the study were professional drivers and office goers followed by students. Women were the least involved.

4. In about 52.6% patients right eye was involved and 22.3% only left eye was involved. In 11.8% both eyes were involved.

5. Time of injury when studied in detail showed that most of the accidents about 42% occurred in night and 30% occurred in morning hours. The remaining 25% occurred in afternoon and evening hours.

6. In this study those involved in RTA were mainly 2 wheelers followed by pedestrians though they were hit mainly by 4 wheelers and 2 wheelers. A few were travelling in autos and 4 wheelers.

7. Though a lot of people were travelling in 2 wheelers, only 15 %were wearing helmets.

8. 60% of people involved in RTA were under the influence of alcohol irrespective of the time of incident though at night it is at the maximum.

9. Regarding fractures involving orbital rim, lateral wall fractures were more common followed by medial, inferior and superior wall fractures. As such about 8% cases had some sort of orbital wall fractures.

10. Among the various periocular signs involved in trauma due to RTA, the most common sign was SCH and Periocular edema, followed by ecchymosis. Various other complications of anterior segment like corneal partial thickness lacerations and full thickness lacerations were observed in the study. Scleral tear, hyphema of various degree, iris prolapse, iridodialysis, traumatic iridocyclitis, pupillary sphincter tear, traumatic cataract, subluxated lens into anterior and posterior chamber were noticed.

11. Posterior segment findings include vitreous hemorrhage, retinal edema, retinal hemorrhages, choroidal rupture, retinal breaks, and retinal detachment. Certain patients after injury may not show any sign but may later develop disc pallor. Patients who develop traumatic optic neuropathy after trauma show severe deterioration of vision with limited posterior segment findings initially. 2 of them improved with steroids while 4 of them did not improve much.

12. Intraocular foreign bodies were found to be in 3.9% and Intraobital foreign bodies were found to be in 2.6%.

13. Grievous injuries:

Corneal lacerartion-3.9%

Traumatic cataract-3.9%

Vitreous hemmorage-3.9%

Retinal detatchment-2.6%

Traumatic optic neuropathy-3.9%

The above grievious injuries noted are more when compared to ocular trauma due to other causes**.**

CONCLUSION

According to this RTA and ocular trauma study conducted at Stanley medical college, ophthalmology department, the following conclusions were made.

The incidence of road traffic accidents is increasing with the increase in vehicle population.

The higher incidence of trauma in men is attributed to the fact that men in our society are exposed to higher level of risk due to RTA since they are more exposed to highway traffic in a heavily populated industrial area like North Chennai.

The age group involved in RTA is becoming younger since younger age groups are using more vehicles especially 2 wheelers.

More RTAs were found to occur during night time when the visibility is poor, awake fullness is less and alcohol abuse is more.

Fractures of orbital rim injuries occur in motorists with severe external injuries. They do occur even with less severe external injuries. Hence there should be a severe suspicion of fracture in all patients with severe pain and defective ocular movements.

Fractures of all types, involving the walls of orbit were noted in RTAs. Fracture of lateral wall of orbit is more common in RTA especially with 2 wheelers.

Those with severe loss of vision should be investigated for direct and indirect causes of traumatic optic neuropathy and those with indirect traumatic optic neuropathy should be given high dose systemic steroids.

Use of helmet:

Of the people driving 2 wheelers, 15% were only wearing helmets, while rests of the 85% patients were not wearing any protective head gears. According to the study patients who were not wearing helmets were more prone for grievous injuries mentioned above.

Almost none of the 3 wheeler and 4 wheeler drivers were wearing seat belts.

Precautionary measures:

Based on the conclusions of the study certain precautionary measures can be undertaken to avoid motor vehicle accidents.

Accidents occur whenever vehicles are more and traffic violations are more. With ever increasing population growth and migration of people to metro cities, government should look into things to decongest the population by creating satellite town ships and creating good job opportunities in sub urban areas.

The Use of public transport system should be encouraged by increasing transport facilities. Vehicle pooling should be advocated, as in certain other countries.

Government should take steps to maintain street lights, reduce power cuts at night, have wider roads with road barricades ,wide medians, separate lanes for different vehicles, more pedestrian crossings, good maintenance of sub ways, maintenance of roads ,filling up of potholes, good water drainage system, creating highways with speed limits, bye-passes and ring roads. Speed monitors are to be introduced in all main roads.

Road lanes should be painted with florescent paints for good visibility during night. Sign boards should be painted with florescent paints.

Stray dogs and cows are main cause of road traffic accidents next to bad roads and use of alcohol. This problem should be addressed along with the blue cross and vetenary department.

Screening the drivers for ocular ailments like refractory errors, diabetes, hypertension, hearing defects should be done periodically especially after 40 years. Use of reflective coated stickers at the back of the vehicles, specific type of horns for specific type vehicles, stickers at the centre of head lamp to avoid glare are to be strictly followed.

Road traffic rules should be a part of the curriculum and school students should be educated to spread the message. The powerful media can be utilized to spread the message. Wearing proper helmets, safety goggles and seat belts should again become compulsory.

After the basic requirements are made available to people, if people are found violating the rules then strict laws should be use to punish the law

breakers. Monitoring traffic at night should be done .Drunken driving should be strictly monitored.

More centers to give first aids and more tertiary centers to treat primarily accidents are to be created. The message of early treatment leading to better outcome should be spread among masses.

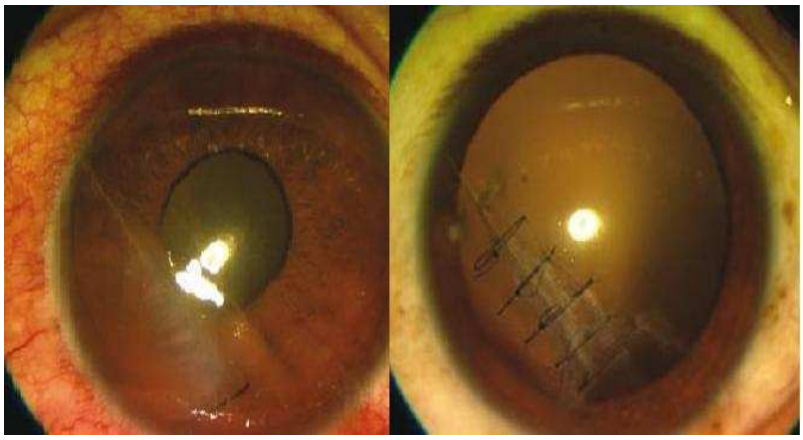
PICTURES

PERIORBITAL EDEMA

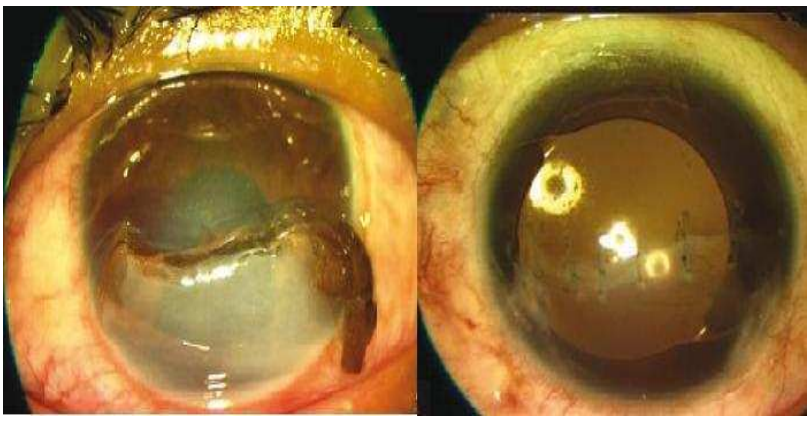
subconjunctival hemorrhage

LID LACERATIONS

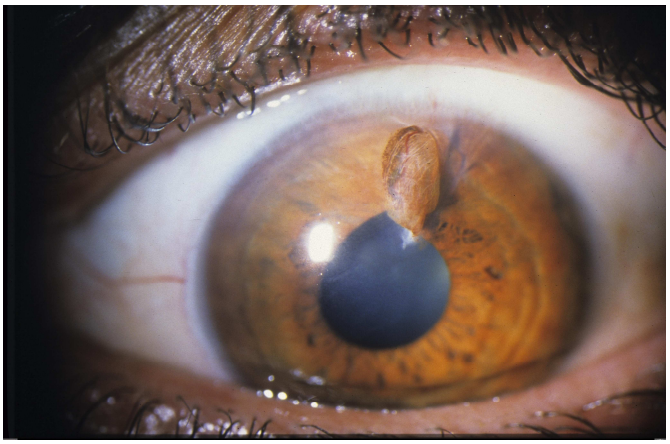
CORNEAL LACERATION-SUTURED

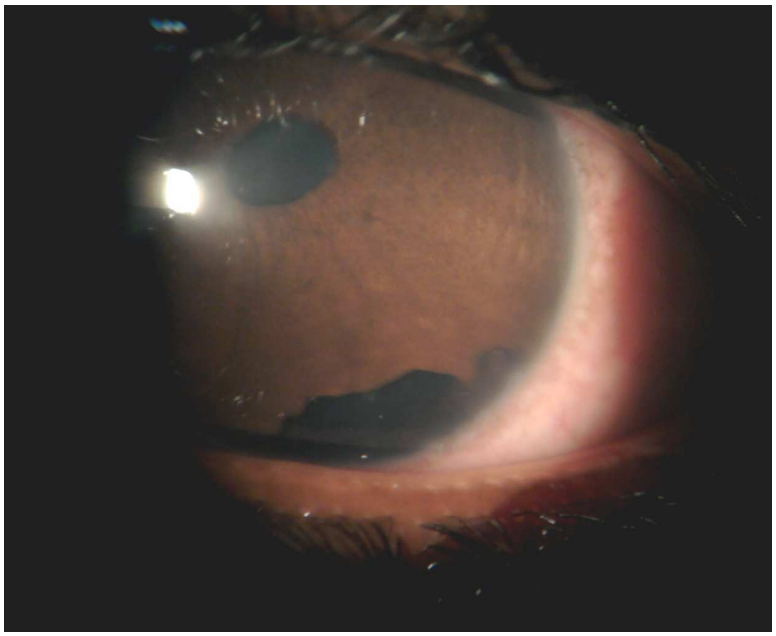


CORNEAL LACERATION WITH IRIS PROLAPSE-(SUTURED)

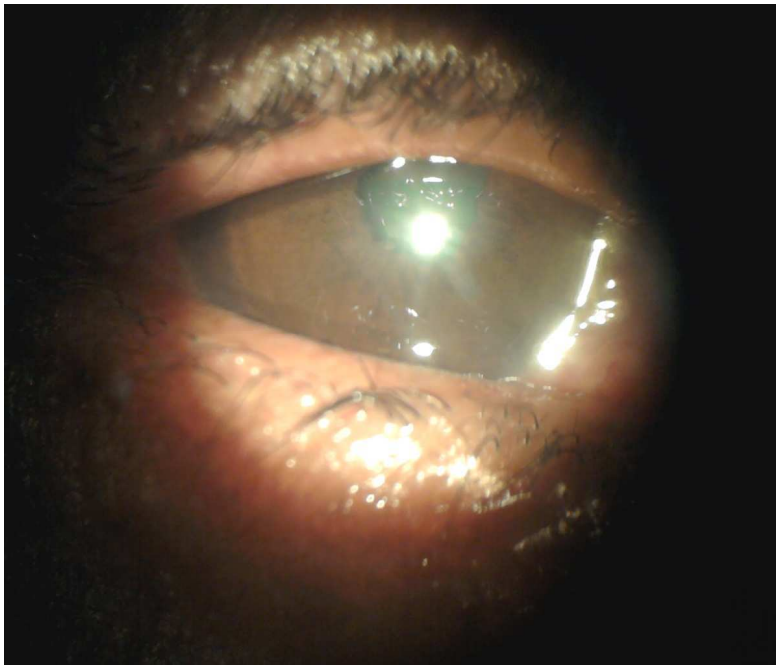


CORNEAL TEAR WITH IRIS PROLAPSE





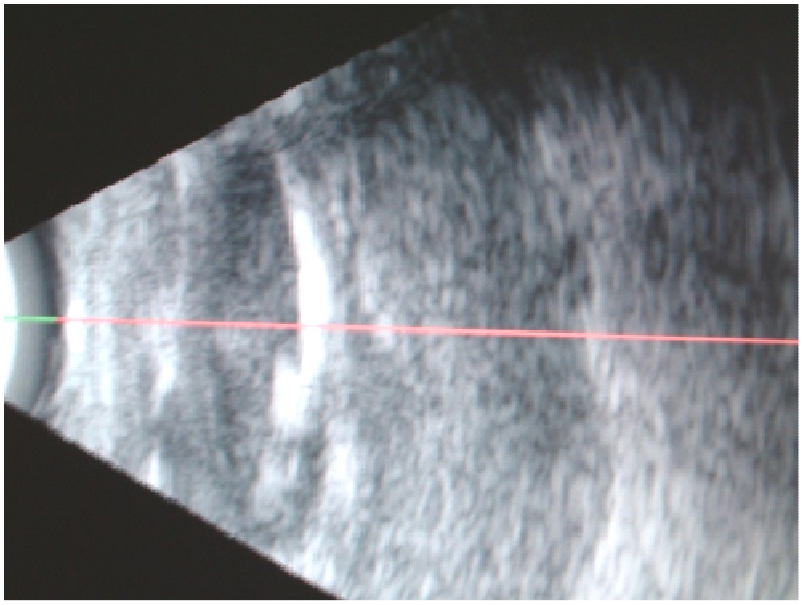
IRIDODIALYSIS



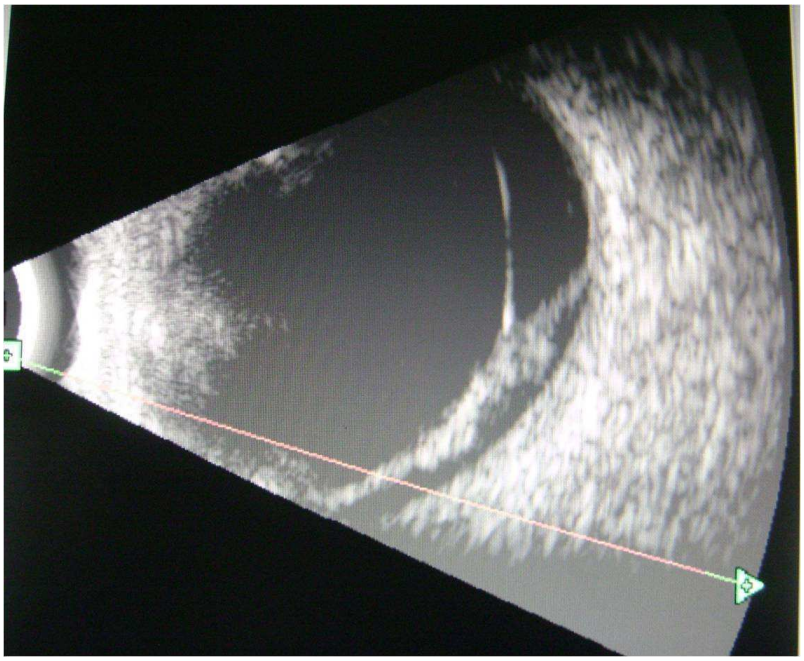
TRAUMATIC MYDRIASIS WITH SPHINCTER TEAR

TOTAL HYPHAEMA

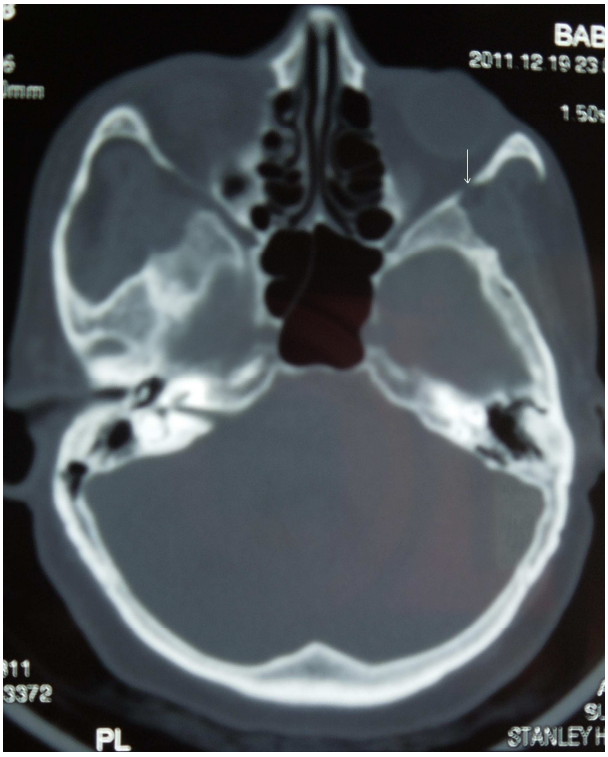
TRAUMATIC CATARACT



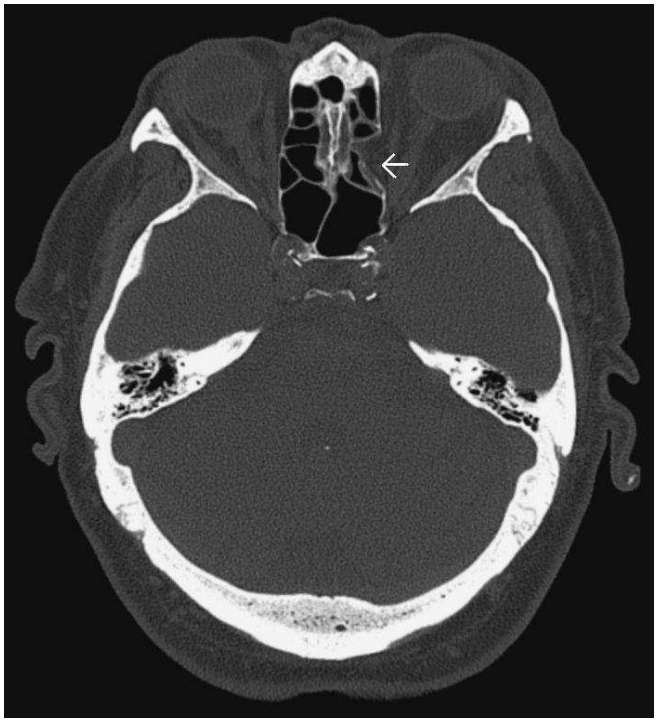
VITREOUS HEMORRHAGE (B SCAN)



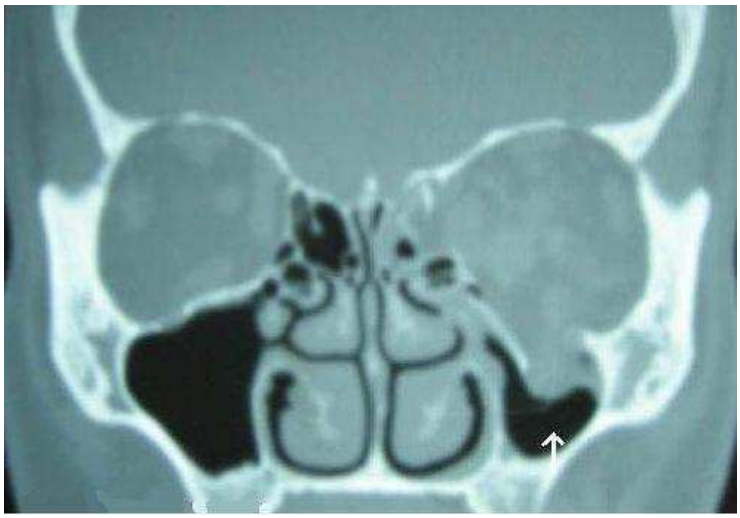
RETINAL DETACHMENT (B SCAN)



LATERAL WALL OF ORBIT FRACTURE



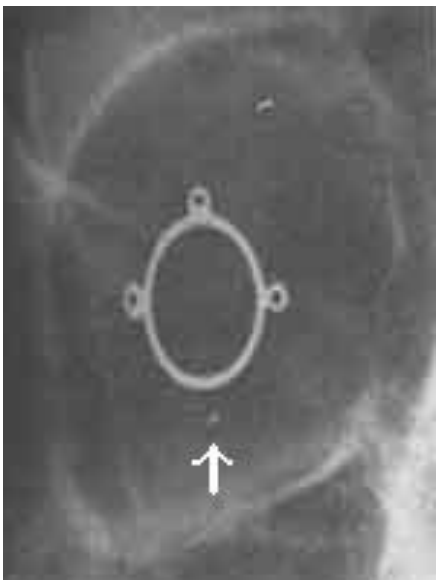
MEDIAL WALL OF ORBIT FRACTURE

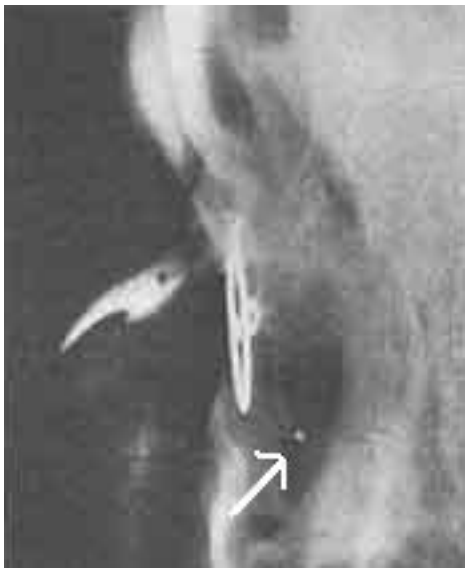


FLOOR OF ORBIT FRACTURE (TEAR DROP SIGN)

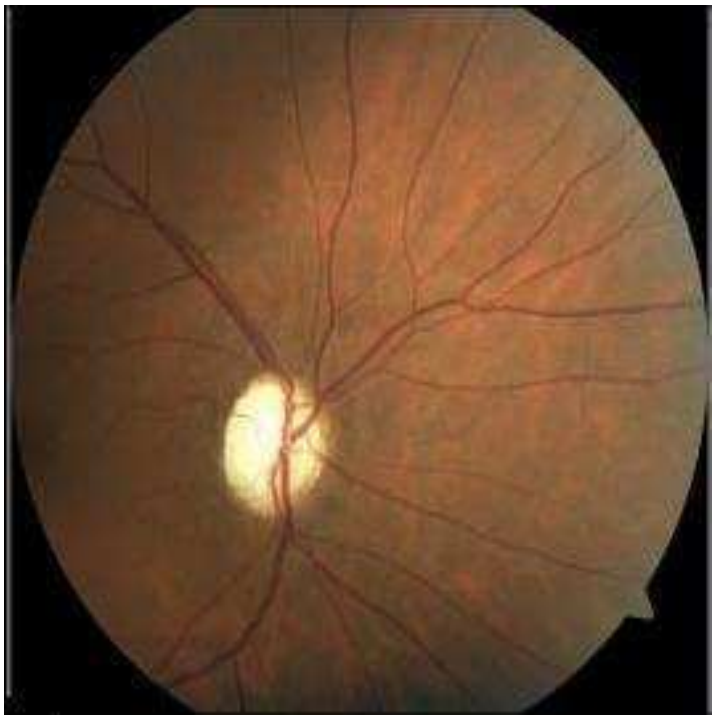
MEDIAL WALL, FLOOR, LATERAL WALL OF ORBIT FRACTURE

INTRA OCULAR FOREIGN BODY

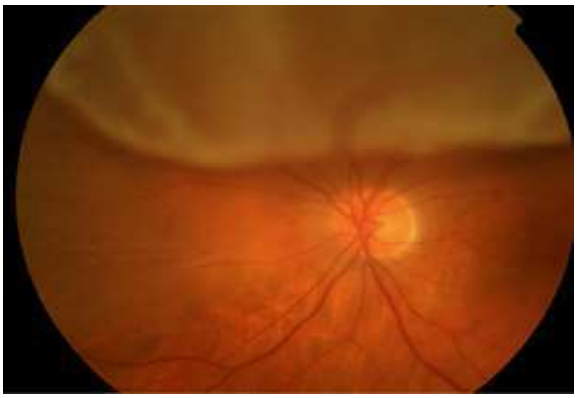




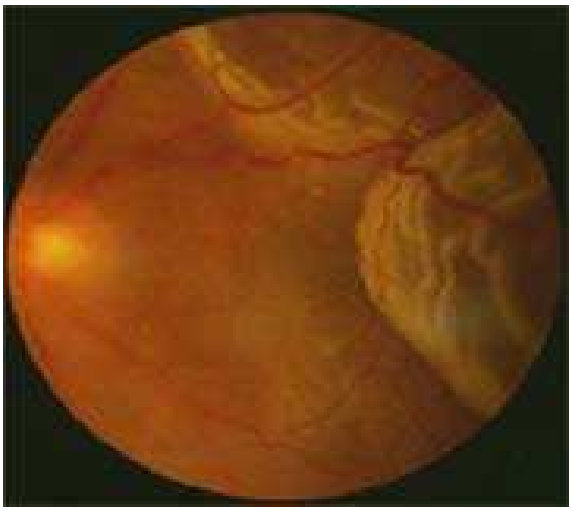
TRAUMATIC OPTIC NEUROPATHY



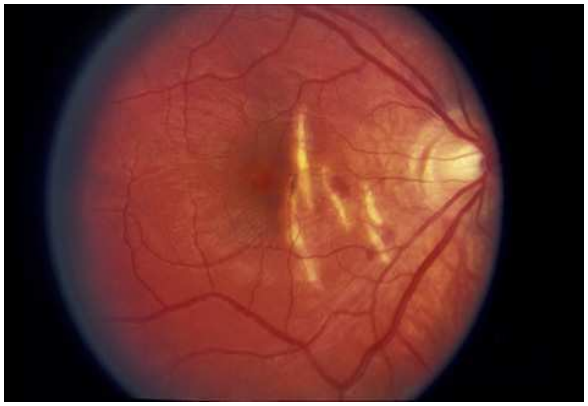
RETINAL DETATCHMENT



RETINAL DETATCHMENT



CHOROIDAL RUPTURE



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PROFORMA

Topic: **Ocular injuries in road traffic accidents- a study**

1. Serial No:

2. Hospital No:

3. Name:

4. Age:

5. Sex: M ☐ F ☐

6. Address:

☐

Rural Urban

7. Occupation: Agriculture Industrial worker drivers Office Student Others

8. Mode of injuries: Pedestrian 2 wheeler 3 wheeler 4 wheeler others

9. Affected eye: Rt ☐ Lt ☐

10. Agent causing injury: Stone Stick Glass Others

11. Date and time of injury:

12. Date first seen at hospital:

13. Nature of first aid given:

14. Ocular status before accident: (by History)

15 H/o alcohol intake victim driver

16. H/O use of Spectacles Helmet Seatbelt

17. Vision: Rt Lt

18 Ocular movements: Full Restricted

19Conjunctiva and Sclera:
20. Cornea:

21 .Anterior chamber:

22 Iris:

23. Pupil:

24. Lens:

25. Posterior segment:
a.vitreous
b.retina
c.choroid

26. Optic nerve:

27. Refraction:

28. Tension:

29. Gonioscopy:

30Investigations: x ray orbit:
X ray skull:
USG: B scan:
CT Orbit -Axial:
-Coronal:
MRI
Others:

31. Diagnosis:

32 Treatment:

33. Follow up: 1month 2month 3month 6month

Master Chart.xls

INDEX TO MASTER CHART

M	-	Male
F	-	Female
OCC	-	Occupation
RE	-	Right Eye
LE	-	Left Eye
BE	-	Both Eye
COR	-	Cornea
CON	-	Conjunctiva
AC	-	Anterior chamber
MYD	-	Mydriasis
VH	-	Vitreous hemorrhage
I	-	Iridocyclitis
#	-	Fracture
S	-	Students
O	-	Office goers
D	-	Drivers
A	-	Agricultural workers
H	-	Home makers.

ON	-	Optic Never
Cho	-	Choroid
Vit	-	Vitreous
Chem	-	Chemosis
Sublux	-	Subluxated Lens

ABSTRACT:

TOPIC: OCULAR INJURIES IN ROAD TRAFFIC ACCIDENTS-A STUDY.

AIM: To study the magnitude and effects of ocular injuries due to road traffic accidents (RTA) and various factors associated with it.

MATERIALS AND METHODS: 175 cases that came as outpatients were included in random fashion and entered in a prestructured profoma.Information regarding time, location, type and mechanism of injury use of spectacles, car safety belts and helmets were obtained.Vision, eye findings, diagnostic tests, medical and surgical treatment were recorded. Whenever necessary x-rays, ultrasonography, CT-scan which the patients had were utilized for the study.152 patients finally included in the study after 6 months follow up.

INCLUSION CRITERIA: 1. Patients who suffered road traffic accidents within 7days.2.Age group>10 to <60 yrs.3.Patients with no previous head injuries.

4.Minimum follow-up is for 6 months.

EXCLUSION CRITERIA: 1.Head injuries and ocular trauma by modes other than RTA.2.Age<1oyrs and>60 yrs.3. Previous history of head and ocular injuries.4.Poor follow up < than 6 months.

RESULTS: About 90% patients were males. 42% were young working people.42% accidents happened during night time.Mostly 2 wheelers were involved in the accidentsand 85% of them were not wearing helmets.60% of patients were under the influence of alcohol.6% had orbital fractures.Lateral wall fractures were more in 2 wheelers riders not wearing

helmets. Various anterior segment findings and posterior segment findings were noted in them. Grievous injuries like corneal laceration, traumatic cataract, vitreous hemorrhage, retinal detachment and traumatic optic neuropathy were found to be more in RTA than due to other causes of trauma.

Conclusion: ocular injuries due to RTA have increased dramatically due to increase in number of vehicles. Young working males, driving at night, alcohol abuse and not wearing helmet are certain risk factors found to be more associated with RTA and ocular injury. Grievous ocular injuries are more in RTA injuries. More 2 wheeler riders were involved in RTAs especially orbital fractures.

Keywords:ocular injury,road traffic accidents.

MASTER CHART

Sl. No	Name	Age	sex	OP/IP No	Occu-pation	helmet	eye inv	drivin g	drun k	Vision		Tension		Anterior Segment							Posterior Segment				Orbit	follow up				
										RE	LE	RE	LE	lid	Conj	Corn	AC	Iris	Pupil	Lens	vit	Retin	Chor	ON		1m	3 m	6m		
1	vinayagan	27	m	36562	S	no	RE	2W	no	6|6	6|6	N	N	Oed/ec	SCH	N	N	N	N	N	N	N	N	N	N	6|6 6|6	6|6 6|6	6|6 6|6		N
2	rajendran	25	m	35328	S	no	LE	2W	y	6|6	6|6	N	N	Oed	SCH	N	N	N	N	N	N	N	N	N	N	6|6 6|6	6|6 6|6	6|6 6|6		A
3	mumtaj	35	f	37272	H	no	RE	3W	no	6|24	6|6			Oed	SCH	edema	I	N	miosed	sublux	VH	hazy/break	hazy	hazy	N	6|12 6|6	6|12 6|6	6|12 6|6		N
4	moorthi	32	m	37945	O	y	RE	2W	no	6|6	6|6	N	N	Oed	SCH/CHE	ab	N	N	N	N	N	N	N	N	N	6|6 6|6	6|6 6|6	6|6 6|6		E
5	jayakanthan	35	m	36406	O	no	LE	2W	no	6|6	6|6	N	N	Oed	SCH	N	N	N	N	N	N	N	N	N	N	6|6 6|6	6|6 6|6	6|6 6|6		N
6	syed	45	m	34978	L	no	LE	2W	no	6|9	6|12	N	N	N	N	N	N	N	N	N	N	N	N	N	N	6|6 6|6	6|6 6|6	6|6 6|6		E
7	satish	22	m	34512	S	no	RE	2W	no	6|12	6|6	N	N	Oed/ec	SCH	N	N	N	N	N	N	N	N	N	N					E
8	mohd.ali	35	m	34587	D	no	RE	2W	y	6|6	6|9	N	N	ode/lac/ec	SCH	N	N	N	N	N	N	N	N	N	N	6|6 6|6	6|6 6|6	6|6 6|6		N
9	abdulla	42	m	38783	O	y	RE	2W	no	6|6	6|6	N	N	Oed	SCH/CHE	ab	I	N	N	N	N	N	N	N	N	6|6 6|6	6|6 6|6	6|6 6|6		N
10	ravi	48	m	34578	O	no	RE	2W	y	6|9	6|9	N	N	N	N	N	I	N	N	N	N	N	N	N	N	6/9,6/9	6|6 6|6	6|6 6|6		N
11	mageshwari	34	f	38737	D	no	RE	2W	no	6|18	6|9	N	N	lac	SCH	N	N	N	N	N	N	N	N	N	N	6|6 6|6	6|6 6|6	6|6 6|6		A
12	ramu	35	m	35456	O	no	RE	3W	no	6|6	6|9	N	N	Oed	SCH	N	N	N	N	N	N	N	N	N	N	6|6 6|6	6|6 6|6	6|6 6|6		N
13	munuswami	35	m	34885	A	no	RE	P	no	1-Jun	6|6	N	N	ode/lac/ec	SCH/CHE	N	N	N	N	N	N	N	N	N	N	6/12,6/6	6|6 6|6	6|6 6|6		N
14	satish kumar	22	m	33523	S	no	LE	2W	no	6|6	6|6	18	20	N	N	N	N	N	N	N	N	N	N	N	N	6|6 6|6	6|6 6|6	6|6 6|6		M
15	shankar	23	m	39385	A	no	LE	4W	y	6/9,	6|6	N	N	Oed/ec	SCH	N	N	N	N	N	N	N	N	N	N	6|6 6|6	6|6 6|6	6|6 6|6		E
16	saravanan	30	m	37749	O	no	RE	2W	no	6|6	6|6	N	N	Oed	SCH	N	N	N	N	N	N	N	N	N	N	6|6 6|6	6|6 6|6	6|6 6|6		M
17	lathif	22	m	37771	S	no	RE	3W	y	6|6	6|6	N	N	Oed/ec	SCH	N	N	N	N	N	N	N	N	N	N	6|6 6|6	6|6 6|6	6|6 6|6		M
18	nehru	32	m	30086	D	y	RE	2W	y	6|60	6|18	N	N	Oed/ec	SCH	N	I	N	miosed	sublux	VH	hazy	hazy	hazy	N	6|36 6|9	6|36 6|9	6|36 6|9	cataract	N
19	ram	40	m	30975	O	no	LE	2W	y	6|6	6|9	N	N	N	SCH	N	N	N	N	N	N	N	N	N	N	6|6 6|6	6|6 6|6	6|6 6|6		M
20	johnson	39	m	39865	A	no	LE	2W	no	6|6	6|6	N	N	ode/lac/ec	SCH/CHE	ab	N	N	N	N	N	N	N	N	N	6|6 6|6	6|6 6|6	6|6 6|6		E
21	karthikeyan	31	m	38452	D	no	BE	P	y	6|12	6|36	N	N	ptosis	SCH	N	N	N	N	N	N	N	N	N	N	6|6 6|6	6|6 6|6	6|6 6|6		A
22	naresh	26	m	35637	O	y	RE	2W	no	6|12	6|6	N	N	Oed	SCH	N	N	N	N	N	N	N	N	N	N	6|6 6|6	6|6 6|6	6|6 6|6		A
23	ramaraj	22	m	37946	S	y	RE	2W	y	6|6	6|6	N	N	N	N	N	N	N	N	N	N	N	N	N	N	6|6 6|6	6|6 6|6	6|6 6|6		E
24	balaji	19	m	37579	S	no	RE	2W	no	6|6	6|9	N	N	N	N	N	N	N	N	N	N	N	N	N	N	6|6 6|6	6|6 6|6	6|6 6|6		M
25	senthamil	23	m	37579	S	no	RE	2W	y	6|9	6|9	N	N	ode/lac/ec	SCH	N	N	N	N	N	N	N	N	N	N	6|6 6|6	6|6 6|6	6|6 6|6		M
26	kalik basha	20	m	38452	D	no	BE	P	y	6|60	6|60	N	N	tear	SCH/CHE	I	I	N	N	N	N	N	N	N	N	6|18 6|12	6|18 6|12	6|36 6|9	cataract	N
27	palavi	40	f	38421	O	no	LE	P	no	6|6	6|9	N	N	Oed	SCH	N	N	N	N	N	N	N	N	N	N	6|6 6|6	6|6 6|6	6|6 6|6		M
28	tamalselvam	23	m	33839	D	no	RE	2W	y	PL+	6|12	N	N	N	SCH	N	I	N	RAPD	sublux	N	break	N	N	lat #	pl+ 6|6	PL,6/6	PL,6/6	RD/paledis	N
29	dhanaselvi	45	f	40838	S	no	RE	3W	no	6|9	6|6	N	N	tear	SCH	N	N	N	N	N	N	N	N	N	N	6|6 6|6	6|6 6|6	6|6 6|6		N
30	munuswami	20	m	42341	S	no	RE	2W	y	6|9	6|9	N	N	Oed/ec	SCH/CHE	N	N	N	N	N	N	N	N	N	floor#	6|6 6|6	6|6 6|6	6|6 6|6		N
31	rajasekar	25	m	44234	S	no	RE	2W	y	6|36	6|243	N	N	Oed	SCH	N	I	N	N	N	N	N	N	N	med #	6|6 6|6	6|6 6|6	6|6 6|6		N
32	srinivasan	40	m	44261	D	no	LE	3W	y	6|12	6|60	N	N	Oed/ec	SCH/CHE	N	N	N	N	N	N	N	N	N		6|6 6|6	6|6 6|6	6|6 6|6		A
33	md.yasar	17	m	44515	S	no	BE	2W	no	6|6	Pldef			tear	SCH	ab	N	D	miosed	sublux	VH	hazy	hazy	hazy	lat/max	6/6,3/60	6/6,6/60	6/6,6/60	pale disc	M
34	prathap	35	m	45683	O	no	RE	2W	y	6|6	6|6	N	N	Oed	SCH	N	N	N	N	N	N	N	N	N	N	6|6 6|6	6|6 6|6	6|6 6|6		E
35	prabakaran	40	m	45150	D	no	RE	2W	no	6|18	6|12	20	18	N	SCH	N	N	N	N	N	N	N	N	N	N	6|6 6|6	6|6 6|6	6|6 6|6		M
36	gajendran	58	m	63601	O	y	LE	3W	y	6|6	6|6	N	N	Oed/ec	SCH	N	N	N	N	N	N	N	N	N	N	6|18 6|12	6|18 6|12	6|18 6|12	cataract	N
37	vadivel	34	m	63780	A	y	LE	2W	y	6|9	6|9	N	N	Oed/ec	SCH	N	N	N	N	N	N	N	N	N	lat#	6|6 6|6	6|6 6|6	6|6 6|6		A
38	rames	40	m	1424	O	no	LE	2W	y	6|12	6|9			ode/lac/ec	SCH/CHE	sen less	N	N	N	N	N	N	N	N	N	6|6 6|6	6|6 6|6	6|6 6|6		M
39	kumar	35	m	13421	A	no	RE	2W	no	6|9	6|6	N	N	Oed/ec	SCH/CHE	N	N	N	N	N	N	N	N	N	N	6|6 6|6	6|6 6|6	6|6 6|6		N
40	sekar	40	m	1887	D	no	BE	4W	y	6|12	PL	N	N	Oed/ec	SCH	N	N	N	RAPD	sublux	N	N	N	atropy	med/lat	6|9 pl+	6|9 pl+	6|9 pl+	pale disc	N
41	chakaravarthi	48	m	2301	O	no	RE	P	no	6|18	6|9	N	N	Oed/ec	SCH	N	N	N	N	N	N	N	N	N	N	6|6 6|6	6|6 6|6	6|6 6|6		M
42	annadurai	45	m	2417	D	no	LE	3W	no	6|9	6|9	N	N	Oed	SCH/CHE	N	N	N	N	N	N	N	N	N	N	6|6 6|6	6|6 6|6	6|6 6|6		E
43	sankar	47	m	2456	A	no	RE	2W	y	6|36	PL			ptosis/ec	tear	edema	shalow	prolapse	miosed	N	N	hazy	hazy	hazy	N	6|6 6|6	6|6 6|6	6|6 6|6		A

44	raguram	42	m	2467	O	no	RE	2W	no	6|60	6|60	N	N	N	SCH	N	N	N	N	N	N	N	N	NN	N	6|6 6|6	6|6 pl+	6|6 pl+		N
45	ekambaram	29	m	3500	D	no	BE	2W	y	6|12	6|18	N	N	Oed	SCH	N	N	N	N	N	N	N	N	N	N	6|6 6|6	6|6 6|6	6|6 6|6		N
46	tangaraj	65	m	3567	A	no	BE	2W	no	6|60	2|60	N	N	ode/lac/ec	SCH/CHE	edema	I	D	myd/tear	N	VH	hazy	hazy	hazy	N	6|24 6|18	6|6 6|6	6|6 6|6		N
47	lilly	29	f	5121	S	no	RE	3W	no	6|9	6|6	N	N	Oed	SCH	N	N	N	N	N	N	N	N	N	N	6|6 6|6	6|6 6|6	6|6 6|6		N
48	gugan	25	m	5404	H	y	LE	2W	no	6|6	6|6	N	N	Oed/ec	SCH/CHE	N	N	N	N	N	N	N	N	N	N	6|6 6|6	6|6 6|6	6|6 6|6		M
49	mari muthu	38	m	5461	O	no	LE	2W	no	6|6	6|6	N	N	Oed	SCH	N	N	N	N	N	N	N	N	N	N	6|6 6|6	6|6 6|6	6|6 6|6		E
50	suresh	28	m	6420	D	no	RE	2W	y	6|12	6|9			tear/ode/ec	SCH/CHE	N	N	N	N	N	N	N	N	N	N	6|6 6|6	6|6 6|6	6|6 6|6		M
51	rahaman	22	m	6894	S	y	LE	2W	no	6|6	PL	N	N	Oed	SCH	N	N	N	N	N	N	N	N		N	6/6.6/60	6/6,6/60	6/6,6/60	paledisc	N
52	cittibabu	30	m	6953	D	no	RE	3W	no	6|12	6|12	N	N	Oed/ec	SCH/CHE	N	N	N	N	N	N	N	N	N	N	6|6 6|6	6|6 6|6	6|6 6|6		M
53	saravanan	25	m	7721	S	no	RE	2W	y	6\36	6|36			tear	SCH/CHE	ab	H	N	myd/tear	sublux	N	N	N	N	N	6|6 6|6	6|6 6|6	6|6 6|6		E
54	vamyal	34	m	7607	D	no	RE	2W	no	6\6	6|6	N	N	Oed	SCH/CHE	N	N	N	N	N	N	N	N	N	N	6|6 6|6	6|6 6|6	6|6 6|6		N
55	sri kumar	40	m	7404	O	no	RE	2W	y	6|60	6|12	N	N	ptosis	SCH/CHE	N	I	D	N	N	VH	hazy	hazy	hazy	N	6|18 6|9	6|18 6|9	6|18 6|9		N
56	sankarraj	18	m	7635	S	y	LE	2W	y	6|6	6|6	N	N	tear	SCH	N	N	N	N	N	N	N	N	N	N	6|6 6|6	6|6 6|6	6|6 6|6		M
57	sankar	26	m	7736	A	no	LE	2W	no	6|6	6|6	N	N	N	N	N	N	N	N	N	N	N	N	N	N	6|6 6|6	6|6 6|6	6|6 6|6		M
58	murugan	32	m	8175	D	no	RE	4W	no	PL+	6|6	N	N	tear	SCH/CHE	N	N	N	RAPD	N	VH	edema	N	atropy	N	pl+ 6|6	pl+ 6|6	pl+ 6|6	pale disc	N
59	venkatesh	31	m	37949	O	no	LE	3W	no	6|9	6|9	N	N	Oed/ec	SCH	N	N	N	N	N	N	N	N	N	N	6|6 6|6	6|6 6|6	6|6 6|6		M
60	dinesh kumar	36	m	8350	A	no	RE	2W	y	6|6	6|6	N	N	Oed	SCH	N	N	N	N	N	N	N	N	N	N	6|6 6|6	6|6 6|6	6|6 6|6		E
61	mahalingam	28	m	8940	A	no	LE	2W	no	6|6	6|9	N	N	ecc	SCH	N	N	N	N	N	N	N	N	N	N	6|6 6|6	6|6 6|6	6|6 6|6		A
62	sabira	46	f	10216	H	no	BE	3W	no	6|12	3|60	N	N	hemato	SCH	N	N	N	RAPD	N	N	N	N	atropy	N	6|12 |5|60	6|12 |5|60	6|12 |5|60	pale disc	N
63	satish	30	m	1165	O	no	RE	2W	y	PL+	6|6	N	N	Oed	SCH/CHE	N	N	N	RAPD	N	N	n	N		N	pl+ 6|6	pl+ 6|6	pl+ 6|6	pale disc	M
64	delli	20	m	11546	S	no	RE	2W	y	6|6	6|6	N	N	N	N	N	N	N	N	N	N	N	N	N	N	6|6 6|6	6|6 6|6	6|6 6|6		A
65	kannan	39	m	11548	O	no	RE	2W	y	6|9	6|9			tear	SCH/CHE	edema	N	N	N	N	N	N	N	N	N	6|6 6|6	6|6 6|6	6|6 6|6		N
66	narayanan	30	m	11758	D	no	LE	2W	no	6|6	6|6	N	N	Oed/ec	SCH/CHE	N	N	N	N	N	N	N	N	N	N	6|6 6|6	6|6 6|6	6|6 6|6		M
67	azhagan	42	m	12847	O	no	RE	2W	y	6|6	6|6	N	N	Oed	SCH/CHE	N	N	N	N	N	N	N	N	N		6|6 6|6	6|6 6|6	6|6 6|6		N
68	kumaran	17	m	11857	S	no	RE	2W	no	6|6	6|6	N	N	Oed/ec	SCH/CHE	N	N	N	N	N	N	N	N	N	floor#	6|6 6|6	6|6 6|6	6|6 6|6		N
69	vinodth	20	m	11870	A	no	BE	P	y	6|9	6|12	N	N	ode/lac/ec	SCH/CHE	N	I	N	myd/tear	N	N	N	tear	N	N	6|6 6|6	6|6 6|6	6|6 6|6		A
70	krishnamorthi	28	m	11856	D	no	RE	2W	no	66|	6|6	N	N	Oed/ec	SCH	N	N	N	N	N	N	N	N	N	N	6|6 6|6	6|6 6|6	6|6 6|6		M
71	shivakumar	44	m	37950	D	no	RE	2W	y	6|12	6|6	N	N	Oed/ec	SCH	N	N	N	N	N	N	N	N	N	ned/floo	6|6 6|6	6|6 6|6	6|6 6|6		N
72	rajesh	20	m	12678	S	no	LE	2W	y	6|6	6|6			Oed	SCH	ab	N	N	N	N	N	N	N	N	N	6|6 6|6	6|6 6|6	6|6 6|6		M
73	santha kumar	20	m	12759	S	no	LE	2W	no	6|6	6|6	N	N	ecc	SCH/CHE	N	N	N	N	N	N	N	N	N	N	6|6 6|6	6|6 6|6	6|6 6|6		E
74	ajith	18	m	12857	S	no	RE	2W	y	6|24	6|12			tear	SCH	N	N	N	N	N	N	N	N	N	N	6|6 6|6	6|6 6|6	6|6 6|6		E
75	ashok kumar	24	m	2737	S	no	RE	2W	y	6|12	6|6	N	N	Oed/ec	SCH	N	N	N	N	N	N	N	N	N	N	6|6 6|6	6|6 6|6	6|6 6|6		M
76	ramesh	34	m	12747	O	no	RE	4W	y	HM	6|9			Oed	tear	lac	I	prolapse	tear	sublux	VH	hazy	hazy	hazy	N	3|60 6|6	3|60 6|6	3|60 6|6	RD	N
77	rathnavel	27	m	36562	S	no	LE	2W	no	6|6	6|6	N	N	Oed/ec	SCH/CHE		N	N	N	N	N	N	N	N	N	6|6 6|6	6|6 6|6	6|6 6|6		M
78	mary	25	f	35328	S	no	LE	2W	no	6|6	6|6	N	N	Oed/ec	SCH	N	N	N	N	N	N	N	N	N	N	6|6 6|6	6|6 6|6	6|6 6|6		M
79	veerasamy	35	m	37272	H	no	RE	3W	no	6|12	6|6			Oed/ec	SCH/CHE	edema	N	N	N	N	N	hazy	hazy	hazy	N	6|6 6|6	6|6 6|6	6|6 6|6		M
80	annadurai	32	m	37945	O	y	RE	2W	y	6|6	6|6	N	N	Oed/ec	SCH/CHE	N	N	N	N	N	N	N	N	N	N	6|6 6|6	6|6 6|6	6|6 6|6		N
81	vignesh	35	m	36406	O	no	LE	2W	no	6|6	6|6	N	N	Oed	SCH	N	N	N	N	N	N	N	N	N	N	6|6 6|6	6|6 6|6	6|6 6|6		N
82	balaram	45	m	34978	L	no	LE	2W	y	6|9	6|12	N	N	N	N	N	N	N	N	N	N	N	N	N	N	6|6 6|6	6|6 6|6	6|6 6|6		N
83	raju	22	m	34521	D	no	RE	4W	y	6|12	6|6	N	N	Oed/ec	SCH/CHE	N	N	N	myd/tear	N	N	N	N	N	N	6|6 6|6	6|6 6|6	6|6 6|6		M
84	balakrishnan	35	m	35123	D	no	RE	2W	y	6|6	6|9	N	N	Oed/ec	SCH/CHE	N	N	N	N	N	N	N	N	N	N	6|6 6|6	6|6 6|6	6|6 6|6		M
85	nagaia	42	m	38783	O	no	RE	P	y	6|6	6|6	N	N	Oed/ec	SCH/CHE	N	N	N	N	N	N	N	N	N	N	6|6 6|6	6|6 6|6	6|6 6|6		M
86	devi sri	48	f	38234	D	no	RE	2W	y	6|9	6|9	N	N	N	SCH	N	N	N	N	N	N	N	N	N	N	6|6 6|6	6|6 6|6	6|6 6|6		E
87	mathy	34	m	38737	D	no	RE	2W	y	6|18	6|9	N	N	lac	SCH/CHE	N	N	N	N	N	N	N	N	N	N	6|6 6|6	6|6 6|6	6|6 6|6		E
88	gunalan	35	m	30121	O	no	RE	3W	y	6|6	6|9	N	N	Oed	SCH/CHE	N	N	N	N	N	N	N	N	N	N	6|6 6|6	6|6 6|6	6|6 6|6		M
89	johnson	22	m	34885	A	no	RE	P	y	6|6	6|6	N	N	Oed	SCH/CHE	N	N	N	N	N	N	N	N	N	N	6|6 6|6	6|6 6|6	6|6 6|6		M

90	ratnakumar	23	m	34581	D	no	RE	2W	y	6|6	6|6	18	19	Oed/ec	SCH/CHE	N	N	N	N	N	N	N	N	N	N	N	6|6 6|6	6|6 6|6	6|6 6|6		E
91	valter	30	m	39385	D	no	LE	4W	y	6|6	6|6	N	N	Oed/ec	SCH	N	N	N	N	N	N	N	N	N	N	N	6|6 6|6	6|6 6|6	6|6 6|6		N
92	kanan	22	m	37123	O	no	RE	2W	y	6|6	6|6	N	N	Oed/ec	SCH/CHE	ab	I	N	N	N	N	N	N	N	N	N	6|6 6|6	6|6 6|6	6|6 6|6		E
93	sangeetha	32	f	3884	H	no	RE	P	no	6|6	6|6	N	N	Oed/ec	SCH/CHE	N	N	N	N	N	N	N	N	N	N	N	6|6 6|6	6|6 6|6	6|6 6|6		M
94	murugan	40	m	30121	O	y	RE	2W	y	6|60	6|18	N	N	ode/lac/ec	SCH	lac	I	prolapse	myd/tear	sublux	N	hazy	hazy	hazy	N	6|36 6|9	6|36 6|9	6|24 6|6	cataract	N
95	santhi	39	f	30975	H	no	LE	P	no	6|6	6|9	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	6|6 6|6	6|6 6|6	6|6 6|6		M
96	taposipal	31	m	39865	D	no	RE	2W	y	6|6	6|6	N	N	Oed	SCH/CHE	N	N	N	N	N	N	N	N	N	N	N	6|6 6|6	6|6 6|6	6|6 6|6		N
97	paul	26	m	36231	D	no	BE	4W	y	6|12	6|36	N	N	ptosis	SCH/CHE	N	N	N	N	N	N	N	N	N	N	N	6|6 6|12	6|6 6|12	6|6 6|12		N
98	murali	22	m	35637	S	no	RE	2W	y	6|12	6|6	N	N	Oed	SCH/CHE	N	N	N	N	N	N	N	N	N	N	N	6|6 6|6	6|6 6|6	6|6 6|6		M
99	sasikumar	19	m	37946	D	y	RE	2W	y	6|6	6|6	N	N	Oed/ec	SCH/CHE	N	N	N	N	N	N	N	N	N	N	N	6|6 6|6	6|6 6|6	6|6 6|6		N
100	siva sanmugam	23	m	37579	O	no	RE	P	y	6|6	6|9	N	N	Oed/ec	SCH	N	N	N	N	N	N	N	N	N	N	N	6|6 6|6	6|6 6|6	6|6 6|6		N
101	murugasan	20	m	37579	S	no	RE	P	y	6|9	6|9	N	N	Oed	SCH	N	N	N	N	N	N	N	N	N	N	N	6|6 6|6	6|6 6|6	6|6 6|6		M
102	gajendran	40	m	37801	O	no	BE	4W	y	6|60	6|60	N	N	ode/lac/ec	SCH	N	I	N	N	N	N	N	N	N	N	N	6|24 6|24	6|24 6|24	6|24 6|24		E
103	vel kumar	23	m	38121	D	no	RE	2W	y	6|6	6|9	N	N	Oed/ec	SCH/CHE	N	N	N	N	N	N	N	N	N	N	N	6|6 6|6	6|6 6|6	6|6 6|6		M
104	ramu	45	m	33839	O	no	RE	P	y	PL+	6|12	N	N	N	SCH	N	N	D	RAPD	N	N	RD	N	N	N	N	pl+ 6|6	pl+ 6|6	pl+ 6|6	LD/pale dis	N
105	satish	20	m	40838	S	no	RE	P	no	6|9	6|6	N	N	ode/lac/ec	SCH/CHE	N	I	N	N	N	N	N	N		N	6|6 6|6	6|6 6|6	6|6 6|6		M
106	dinesh kumar	25	m	42341	S	no	RE	2W	no	6|9	6|9	N	N	Oed	SCH/CHE	N	N	N	N	N	N	N	N	N	N	6|6 6|6	6|6 6|6	6|6 6|6		E
107	vinoth	40	m	44234	O	no	RE	P	y	6|36	6|24	N	N	Oed/ec	SCH/CHE	lac	N	N	myd/tear	N	VH	N	N		N	6|12 6|12	6|12 6|12	6|12 6|12		M
108	babu	17	m	44261	A	no	LE	3W	no	6|12	6|60	N	N	Oed	SCH/CHE	N	H	N	N	N	N	N	N	N	N	6|9 6|24	6|9 6|24		retinalbreal	N
109	kuppamal	35	m	44515	D	no	BE	P	y	6|6	Pldef			tear	SCH/CHE	N	N	N	RAPD	N	N	N	N	N	N	6|6 2|60	6|6 2|60	6|6 2|60	paledisc	N
110	prema	40	f	45683	H	no	LE	2W	no	6|6	6|6	N	N	Oed	SCH/CHE	N	N	N	N	N	N	N	N	N	N	6|6 6|6	6|6 6|6	6|6 6|6		M
111	bhuvaneshwari	58	f	45150	H	no	RE	2W	no	6|18	6|12	20	18	N	SCH	N	N	N	N	N	N	N	N	N	N	6|6 6|6	6|6 6|6	6|6 6|6		E
112	girija	34	f	63601	H	no	BE	P	no	6|6	6|6	N	N	Oed/ec	SCH/CHE	N	N	N	N	N	N	N	N	N	N	6|6 6|6	6|6 6|6	6|6 6|6		N
113	srinivasan	40	m	63780	D	no	LE	P	y	6|9	6|9	N	N	Oed/ec	SCH/CHE	ab	N	N	N	N	N	N	N	N	N	6|6 6|6	6|6 6|6	6|6 6|6		M
114	balaia	35	m	1424	D	no	LE	2W	y	6|12	6|9	19	20	Oed/ec	SCH	sen less	N	N	N	N	N	N	N	N	N	6|6 6|6	6|6 6|6	6|6 6|6		M
115	madivanan	40	m	13421	D	no	RE	2W	y	6|9	6|6	N	N	Oed/ec	SCH/CHE	N	N	N	N	N	N	N	N	N	N	6|6 6|6	6|6 6|6	6|6 6|6		N
116	samson	48	m	1887	D	no	BE	2W	y	6|12	PL	N	N	Oed/ec	SCH/CHE	N	I	N	RAPD	N	N		N		lat #	6|9 pl+	6|9 pl+	6|9 pl+	paledisc	N
117	nagarajan	45	m	2301	O	no	RE	2W	y	6|18	6|9	N	N	Oed	SCH/CHE	N	I	N	N	N	N	N	N	N	N	6|6 6|6	6|6 6|6	6|6 6|6		M
118	sasikumar	47	m	2417	D	no	BE	P	y	6|9	6|9	N	N	Oed/ec	SCH	N	N	N	N	N	N	N	N	N	N	6|6 6|6	6|6 6|6	6|6 6|6		M
119	bala	42	m	2456	A	no	LE	P	y	6|36	PL	N	N	ptosis	tear	edema	shall	Prolpse/D	tear	N	N	hazy	N	N	N	6|12 2|60	6|12 2|60	6|12 2|60	etinalbreal	N
120	kattan	29	m	2467	D	no	RE	P	y	6|60	6|60	N	N	N	N	N	H	N	N	N	N	N	N	N	N	6|24 6|24	6|24 6|24	6|24 6|24		M
121	sasidaran	65	m	3500	S	no	BE	2W	no	6|12	6|18	N	N	ode/lac/ec	SCH/CHE	N	N	N	myd/tear	N	N	N	N	N	N	6|6 6|6	6|6 6|6	6|6 6|6		M
122	marayappan	29	m	3567	A	no	BE	2W	y	6|60	2|60	N	N	ode/lac/ec	SCH/CHE	edema	N	N	N	N	N	N	hazy	hazy	N	6|36 4|60	6|36 4|60	6|36 4|60	cataract	N
123	sidambaram	25	m	3890	O	no	RE	P	y	6|9	6|6	N	N	Oed	SCH	N	N	N	N	N	N	N	N	N	N	6|6 6|6	6|6 6|6	6|6 6|6		M
124	ravi	38	m	5404	O	no	LE	2W	no	6|6	6|6	N	N	Oed/ec	SCH/CHE	N	N	N	N	N	N	N	N		N	6|6 6|6	6|6 6|6	6|6 6|6		N
125	anand	28	m	5461	D	no	LE	2W	no	6|6	6|6	N	N	Oed	SCH/CHE	N	N	N	N	N	N	N	N	N	N	6|6 6|6	6|6 6|6	6|6 6|6		N
126	ramalingam	22	m	6420	S	no	RE	2W	y	6|12	6|9			ode/lac/ec	SCH/CHE	ab	N	N	N	N	N	N	N	N	N	6|6 6|6	6|6 6|6	6|6 6|6		M
127	suresh	30	m	6894	O	y	RE	2W	y	6|6	6|6	N	N	Oed/ec	SCH	N	N	N	N	N	N	N	N	N	N	6|6 6|6	6|6 6|6	6|6 6|6		N
128	chandru	25	m	6953	A	no	BE	P	y	6|12	6|12	N	N	ode/lac/ec	SCH/CHE	N	I	N	N	N	N	N	N	N	N	6|6 6|6	6|6 6|6	6|6 6|6		M
129	rani	34	m	7721	H	no	RE	2W	y	6\36	6|36			tear	SCH	lac	N	N	N	N	N	N	N	N	N	6|6 6|6	6|6 6|6	6|6 6|6		N
130	prabhu	40	m	7607	A	no	RE	2W	y	6\6	6|6	N	N	Oed/ec	SCH	N	N	N	N	N	N	N	N	N	N	6|6 6|6	6|6 6|6	6|6 6|6		E
131	ramesh	18	m	7404	S	no	RE	P	no	2|60	6|12			ptosis	SCH	ab	H	N	myd/tear	N	N	N	N	N	N	3|60 6|6	3|60 6|6	3|60 6|6	RD	N
132	govind	26	m	7635	S	no	RE	2W	no	6|6	6|6			ode/lac/ec	SCH/CHE	N	N	N	N	N	N	N	N	N	N	6|6 6|6	6|6 6|6	6|6 6|6		E
133	mahendran	22	m	7736	D	no	RE	2W	y	6|6	6|6	N	N	oed	SCH	N	N	N	N	N	N	N	N	N	N	6|6 6|6	6|6 6|6	6|6 6|6		N
134	jayaraman	31	m	8175	A	no	RE	2W	y	PL+	6|6	N	N	tear	SCH	N	N	N	N	N	N	N	N	N	max#	pl+ 6|6	pl+ 6|6	pl+ 6|6	etinalbreal	M
135	indran	36	m	8213	D	no	LE	P	no	6|9	6|9	N	N	ode/lac/ec	SCH/CHE	N	N	N	N	N	N	N	N	N	N	6|6 6|6	6|6 6|6	6|6 6|6		E

136	balaji	28	m	8350	O	no	RE	2W	no	6|6	6|6	N	N	Oed/ec	N	N	N	N	N	N	N	N	N	N	N	N	6|6 6|6	6|6 6|6	6|6 6|6		N
137	venkatesh	46	m	8940	D	no	LE	2W	y	6|6	6/	N	N	ecc	SCH	N	N	N	N	N	N	N	N	N	N	N	6/6,6/24	6|6 6|24	6|6 6|36	cataract	E
138	dayalan	30	m	10216	D	no	BE	P	y	6|12	6|60	N	N	hemato	SCH	N	I	N	N	N	N	N	N	N	N	N	6|6 6|6	6|6 6|6	6|6 6|6		N
139	yasmin	28	f	10314	O	no	RE	2W	y	PL+	6|6	N	N	Oed	SCH/CHE	N	N	N	myd/tear	N	N	N	N	N	N	1|60 6|6	1|60 6|6	1/60,6,9	RDCatar	N
140	tamilselvam	39	m	11546	S	no	RE	2W	no	6|6	6|6	N	N	oed	SCH	N	N	N	N	N	N	N	N	N	N	N	6|6 6|6	6|6 6|6	6|6 6|9		E
141	gandhi	30	m	11548	O	no	RE	2W	y	6|9	6|9	N	N	ode/lac/ec	SCH/CHE	N	N	N	N	N	N	N	N	N	N	N	6/12,6/12	6/12,6/6	6/18,6/6	chrupture	E
142	akbar	42	m	11758	D	no	RE	2W	no	6|6	6|6	N	N	Oed/ec	SCH/CHE	N	N	N	N	N	N	N	N	N	N	N	6|6 6|6	6|6 6|6	6|6 6|6		N
143	basha	17	m	12847	O	no	RE	2W	y	6|6	6|6	N	N	Oed	SCH	N	N	N	N	N	N	N	N	N	N	N	6|6 6|6	6|6 6|6	6|6 6|6		N
144	rajesh	20	m	11857	D	no	RE	3W	y	6|6	6|6	N	N	Oed/ec	SCH	N	N	N	N	N	N	N	N	N	N	N	6|6 6|6	6|6 6|6	6|6 6|6		N
145	martamma	28	f	11870	H	no	BE	2W	no	6|9	6|12	N	N	ode/lac/ec	SCH/CHE	ab	N	N	N	N	N	N	tear	N	lat#	6|6 6|6	6|6 6|6	6|6 6|6		N
146	lakshmanan	44	m	11001	O	y	RE	P	y	#####	6|6	N	N	Oed	SCH	N	N	N	N	N	N	N	N	N	N	N	6/24,6/6	6/24,6/6	6/24,6/6	cataract	E
147	lakshmannan	20	m	37950	D	no	RE	2W	y	6|12	6|6	N	N	ecc	SCH/CHE	N	I	N	N	N	N	N	N	N	N	N	6|6 6|6	6|6 6|6	6|6 6|6		E
148	shivakumar	18	m	12901	S	no	LE	2W	y	6|6	6|60	N	N	Oed	SCH	lac	N	prolapse	tear,rapd	N	N	N	N	N	N	6|6 6|36	6|6 6|36	6|6 6|17	RD,paledis	N
149	elumalai	24	m	11234	A	no	RE	P	y	6|6	6|6	N	N	Oed/ec	SCH	N	N	N	N	N	N	N	N	N	N	N	6|6 6|6	6|6 6|6	6|6 6|18		E
150	srinivasan	34	m	12857	A	no	RE	P	y	1|60	6|12	N	N	ode/lac/ec	SCH/CHE	ab	H	N	myd/tear	N	N	N	N	N	N	2|6o 6|9	2|6o 6|9	6|6 6|19	etinalbreal	N
151	karuppu	23	m	12212	A	no	RE	P	no	6|12	6|6	N	N	Oed/ec	SCH	N	N	N	N	N	N	N	N	N	N	6/12.6/6	6/18,6/6	6/24,6/6	ch0roidalte	E
152	RAJAsekar	40	m	12500	A	y	RE	P	no	6|12	6|6	N	N	Oed/ec	N	ab	N	N	N	N	N	N	N	N	N	6|6 6|6	6|6 6|6	6|6 6|6		N